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AUTONOMIC DYSFUNCTION IN THE INNER EAR.*

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The autonomic nervous system is the mediator in the body's adjustment to its environment. The character of one's autonomic system is a matter of inheritance. There is variation in the quality and performance of the system in different individuals. This fact has been a matter of medical record since early times and was given particular impetus by the writings of Eppinger and Hess,[1] who, in 1909, introduced the clinical pictures of the sympatheticotonic individual and the vagotonic individual into medical literature. It is interesting to follow the emergence of the autonomic system from the anatomic awareness of the nineteenth century to its present position in the forefront of functional clinical medicine. Diseases then recognized and described only in the textbooks of morbid anatomy are now recognized and intercepted in the stage of early altered physiology — the stage in which autonomic dysfunction has not yet been translated into permanent tissue change. The era of bacterial supremacy in disease concepts appears to be giving way to a realization that intrinsic bodily dysfunction in the neurovascular, neurosecretory and neuromuscular mechanisms may be of more importance in many previously obscure disorders than the presence of bacteria of dubious pathogenicity. The focal disease theory shrinks into relative insignificance as one bodily disorder

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after another, previously ascribed to it, is demonstrated to result from autonomic dysfunction. The emphasis of recent years on psychosomatic medicine is an expression of general medical awareness of the impact of man's environment on a neural mechanism which may not be equal to a smooth performance of its task.

The inner ear is a structure well designed anatomically to give gross symptomatic expression to very minor alterations in its neurovascular and, perhaps, neurosecretory mechanisms. Few end-organs in the body are as expressive of minute changes of autonomic control. The inner ear is a rigidly closed space. Circulation is supplied by an end-artery and its branches. Collateral circulation is absent. The neural mechanisms dependent upon this arterial supply are minute and delicate. Extremely fine vascular change results in relatively severe subjective sensory disturbance. These may be of an acoustic nature, a static nature, or both, dependent upon the arterial branch involved.

The main artery is the internal auditory, a branch of the basilar. Its ramifications supply all parts of the labyrinth. A noteworthy branch, the vestibulocochlear artery, carries blood to the basal turn of the cochlea and to a portion of the saccule, utricle and posterior ampulla of the vestibule. Vascular tonus in these arterial branches and their arteioles is controlled by the autonomic innervation of the inner ear.

The autonomic nervous system is a motor mechanism comprised in reality of two opposed systems which maintain the equilibrium of the organism's adjustment to its environment — the one is the sympathetic system, the other the parasympathetic. Sympathetic innervation of the labyrinth arises from the thoracic cord and extends cephalad in the cervical sympathetic trunk and its continuation, the internal carotid nerve. The inferior caroticotympanic branch of the latter traverses the bony septum between the carotid canal and middle ear. It is here joined by a branch of the IXth cranial nerve, which may contain parasympathetic elements, and the ramifications of the two on the promontory of the middle ear are termed the tympanic plexus.

The tympanic nerve which emerges from the tympanic plexus traverses the middle fossa surface of the petrous portion of the temporal bone as the lesser superficial petrosal nerve. By connections of the latter with the geniculate ganglion, as well as by direct connecting fibres from the tympanic plexus to the geniculate ganglion and VIIth nerve, sympathetic fibres from the cervical trunk and possible parasympathetic fibres from the IXth cranial nerve reach the VIIth. The VIIIth nerve, or cochleovestibular nerve, carries few autonomic nerve fibres from the brain stem. Yet, as shown by Lorente de No,[2] this nerve does have such fibres when it ramifies to the cochlear and vestibular end-organs in the fundus of the internal auditory canal. A connecting nerve filament which carries these autonomic fibres from the VIIth to the VIIIth cranial nerve in their course in the internal auditory canal was demonstrated by de No in 1925. It completes the anatomic basis for the neurovascular syndromes of the inner ear.

There is little known about the effect of increased parasympathetic tonus in the inner ear. It is possible that it controls neurovascular phenomena through vasodilator activity and produces common clinical pictures. The usual experimental study has been done, not by stimulation of the parasympathetic as a dynamic vasodilating force, but rather by destruction of the sympathetic innervation to produce that effect passively. Direct sympathetic stimulation has been carried out repeatedly. A mass of experimental work has followed three general lines:

1. Cervical sympathectomy and stimulation of the sympathetic at the cervical trunk or below.
2. The effects of drugs acting on the autonomic system.
3. The results of operations upon man with and without ear disease.

Fraser[3] has summarized the work:

"The results are somewhat conflicting, but the conclusion which emerges is as follows: constriction of the labyrinthine

vessels leads to an increase in the excitability of the vestibule. Dilatation of these vessels leads to a diminution in the excitability of the vestibule with an increase in the acuity of hearing." The important clinical fact is that the changes in vascular tonus and the resultant changes in fluid balance within the endolymphatic system are produced by dynamic forces and not by static influences of ionic concentrations through semipermeable membranes.

One inherits his autonomic nervous system. If it be an unstable one, the potentiality toward certain clinical disorders is great. The impact of the environment results in manifestations of autonomic imbalance in many body systems. The mechanism is sometimes set in motion by single, specific effectors, such as foods or inhalant substances and the body system involved becomes established as a shock organ which thereafter may react to those substances independent of central neural controls. The clinical picture of allergy results. Often, and particularly in a vigorous climate, physical factors of temperature and humidity and their rapid alteration will produce these striking autonomic effects. The result has been spoken of as physical allergy and sometimes as intrinsic allergy.

There remains yet another effector, and more important perhaps than extrinsic food, inhalant or physical factors, which can produce marked clinical disorder through autonomic disturbance. That effector is the individual's own emotion. This factor brings confusion to the ordinary concepts of allergy. It is the effector in a high percentage of the neurovascular, neurosecretory and neuromuscular reactions which make up the picture of the intractable, or fixed allergic.

Autonomic dysfunction in the inner ear comes under the influence of these effectors. In the same individual, one or all may be capable of producing the clinical syndromes of the inner ear. Foods and inhalants are of least importance. The inner ear is not commonly their shock organ. The individual who develops tinnitus, vertigo and hearing impairment from a specific food or inhalant is uncommon enough to be worthy of remark. Physical factors have more significance. Tinnitus

is more commonly noted during periods of inclement weather and changes of season. Vestibular vertigo, with or without cochlear symptoms, is more frequently seen during the same periods. These, however, are observations that are difficult to demonstrate statistically because the individual who is truly physically sensitive is usually also most subject to emotional effects through the same autonomic mechanism, and emotional stress knows no season.

Williams[4] organized the many autonomic imbalances of the head into the picture of intrinsic allergy of the head. One patient may manifest neurovascular and neurosecretory irregularities in many systems. In the head, Williams has grouped these into myalgias, vasodilating pains, chronic vasomotor rhinitis and endolymphatic hydrops.

The terminology of the inner ear syndromes is confusing. It has run the gamut from Ménière's disease to endolymphatic hydrops and from catarrhal deafness to endolymphatic hydrops without vertigo. In order that all degrees of autonomic dysfunction in the inner ear be given a common etiologic term, it would seem preferable to denote the disturbance as vasomotor labyrinthitis where all components of the inner ear are involved, and vasomotor cochleariti or vasomotor vestibulitis where only acoustic or only static components have yet been involved.

The changes in neurovascular tonus may be precipitate and extreme or they may be of minor degree and long continued. The cataclysmic disorder of major neurovascular involvement was organized into a syndrome by the writings of Ménière. The chronic progressive degeneration of long continued minor neurovascular change has been obscured as catarrhal deafness or primary VIIIthe nerve neuritis for several medical generations. The altered physiology producing both pictures differs not in character but in degree.

Careful history demonstrates that in 80 per cent of patients a premonitory phase precedes a first violent inner ear attack. This phase may last for years and be characterized only by intermittent tinnitus, with or without gradually developing

hearing impairment, or by mild, transient vertigo, elicited only by abrupt positional change. These symptoms are evidences of poor autonomic adjustments in the neurovascular control of the inner ear. At this stage of the disorder the majority of patients will evidence other symptoms of cranial autonomic instability, in the form of myalgia, vasodilating pain or chronic vasomotor rhinitis, or in a positive family history of similar parental or sibling involvement. In fact, the associated vasomotor rhinitis and pain phenomena have been so commonly noted in association with these ear symptoms in past decades that the misnomer of catarrhal deafness became descriptive of the chronic progressive cochlear degeneration which resulted from vasomotor cochleariti s. The inept practice of auditory tubal inflation became a standard otologic procedure. It is embarrassing to reflect today on the hundreds of thousands of middle ear inflations in which otologists have doggedly indulged in an effort to relieve by aerodynamics a disorder which in reality results from neurovascular disturbance in the inner ear.

Most patients with inner ear symptoms from faulty autonomic balance never manifest the complete syndrome of inner ear disruption. All are potential candidates, however. The first appearance of tinnitus or of vertigo on quick alteration of position in a patient with associated evidence of autonomic imbalance or pronounced family history of same marks the individual in the astute clinician's mind. It is interesting to follow the vasomotor vagaries of one such individual for several years and see the first complete inner ear syndrome appear suddenly and cataclysmically after a particularly severe environmental stress. For every individual who progresses to this point there are scores who continue for years with lesser involvements. When the latter concern vestibular arterial branches only, the pseudo-Ménière's syndrome of Dandy or vasomotor vestibulitis is noted in varying degrees of severity. When cochlear arterial segments alone are involved the vasomotor cochleariti s results in intermittent or constant tinnitus and progressive and permanent inner ear type hearing impairment. If the vestibulocochlear artery from

the internal auditory is affected, tinnitus of high pitch, high tone impairment and variable vertigo result.

The pathological change in the inner ear which results from vascular alteration can conceivably take two forms; that resulting from frequent minor changes in vessel lumina and that resulting from sudden major alteration in circulation. These may differ not at all in mechanism of production but only in degree of involvement. Relative to the former, the observations of Guild[5] on the pathology of chronic progressive inner ear deafness are of moment. He states:

"Normal looking blood vessels are present in our sections in regions with total atrophy of the organ of Corti. The fact that the areas of end-organ atrophy are often very sharply circumscribed suggests occlusion of terminal arteries as the cause; we have no histological evidence, however, to support this view. The clinical and pathological evidences of systemic vascular disease, as recorded in our tables, also indicate that arteriolosclerosis is not the cause of organ of Corti atrophy." It is probable that recurrent and transient alterations in vessel lumina produce the permanent neural damage without histologically demonstrable vessel change.

At the other end of the scale of neurovascular change, the pathology of the complete inner ear syndrome with violent clinical manifestations in acoustic and static spheres was shown first by Hallpike and Cairns[8] and since corroborated by Lindsay[9] and many others. They have shown a gross dilatation of the endolymphatic system with destruction of the neural elements. This gross transudation and edema can only have formed through dynamic vasomotor change.

The mechanism whereby autonomic dysfunction produces these changes is not known. In the vasomotor unstable individual of this type, Muller[6] described an inherited tendency for capillary loops to behave in an abnormal manner on exposure of the organism to physical and emotional stimuli. Spasm at the arteriolar end of the capillary results in endothelial change with increase in permeability and the formation of edema. The observations of Abell and Schenck[7] have con-

tributed further support to the arteriolar spasm theory of edema fornation. These changes can be mediated through autonomic innervation.

Drugs possessing the ability to block central autonomic influence have favorable effect on vasomotor changes in the inner ear. Among these, tetraethylammonium salts are relative newcomers. When they are used intravenously or intramuscularly a transient autonomic block at ganglion level is established and it can terminate an acute severe vasomotor labyrinthine attack.

Modern therapy in vasomotor cochleariti, vasomotor vestibulitis and vasomotor labyrinthitis, in either the acute or chronic form, aims at re-establishing the integrity of the inner ear circulation. This may be accomplished through the neural blocking influence of tetraethylammonium chloride or the antihistamine drugs, or more directly through the vigorous vasodilating action of intravenous histamine or oral nicotinic acid. There is no completely satisfactory drug available to us yet.

Neural damage resulting from ischemia quickly becomes irreversible. Treatment of the acute vasomotor crisis in the inner ear is emergent. Otologists have an important education task before them in conveying that knowledge to the general medical group and to the public. Neural damage from chronic vasomotor change is always irreversible. Prevention is our only cure. It is important to recognize the vasomotor unstable individual and demonstrate his early loss audiometrically. Nicotinic acid is the best drug yet available for long term usage.

Drug therapy has distinct limitations in a disorder in which emotional factors play a major etiologic rôle. The patient with early and still tractable vasomotor irregularity in his labyrinth often needs correction in his attitude toward life. Drug therapy serves best in the acute severe phase of vasomotor change. Psychotherapy is frequently more important in maintaining stability once regained. This is a fact which cannot be sufficiently emphasized. The otologist, like any doctor of medicine, must fill a psychiatric rôle.

Some systemic factors have an influence on the production of inner ear symptoms through autonomic mediation. In the treatment of vasomotor change in the inner ear they should be given proper attention as contributors. The endocrine system is controlled by the autonomic nervous system and the hypophysis. Endocrine factors in women which influence vasomotor control during the menopause or at the time of the menstrual period accent vertigo and tinnitus in the autonomic unstable individual during those periods. The low metabolic rate commonly found in both males and females with autonomic instability need not be a cause of their inner ear symptoms, but rather another result of faulty autonomic regulation. The use of thyroid, therefore, should not be viewed as a complete method of treatment, but as an adjunct only. The influence of fatigue and tobacco upon the vasomotor system makes adequate rest and abstinence from smoking one of the first rules of proper therapy.

The patient with far advanced vasomotor labyrinthitis usually has permanent loss of useful hearing in the involved side with a vestibular mechanism still sufficiently viable to repeatedly disable him with attacks of vertigo. Often his abnormal neurovascular habit is so deeply rooted that it responds poorly, if at all, to medical and psychiatric correction. The number of advanced cases comprises an extremely small per cent of the total with vasomotor labyrinthine disease. With early recognition and proper medical and psychiatric treatment, this small per cent will dwindle further. Surgical destruction of the involved labyrinth is the only control measure one can at present offer the intractable case.

Present-day labyrinthotomy destroys the useful residual hearing on the operated side. For that reason, this surgery is reserved for disabled and uncontrollable cases with no useful hearing remaining on the involved side. The possibility of future opposite side involvement in so general a disorder as vasomotor instability exists. The sacrifice of useful hearing could be disastrous if, in the future, vasomotor cochlearitis developed in the opposite ear.

The use of streptomycin by intramuscular injection to

destroy the vestibular end-organ by selective neurotoxic action is receiving wide attention. It is possible that its usage should be limited until its neurotoxicity is conclusively shown to exist for the vestibular end-organ alone. An infrequent case of vasomotor vestibulitis may suffer severe and disabling vertigo, yet show no vasomotor change in the cochlea. Labyrinthotomy is not applicable because useful hearing would be destroyed. Streptomycin destruction of the vestibules would seem reasonable in such an instance.

Summary: The syndromes of the inner ear result from neurovascular and possibly neurosecretory change in the labyrinth.

This change may be acute and severe as the result of sudden alteration in the vascular tonus of large segments of the neural mechanism of the inner ear and produce the picture of severe acute vasomotor labyrinthitis, vasomotor cochleariitis or vasomotor vestibulitis, or it may be chronic and insidious as the result of long continued and intermittent alterations in the vascular tonus of many small segments of the neural mechanism of the inner ear and result in chronic vasomotor labyrinthitis, vasomotor cochleariitis or vasomotor vestibulitis.

These changes are mediated by autonomic mechanisms and differ not in kind but in degree.

They occur most frequently in individuals with inherited autonomic instability.

The majority of these individuals manifest evidences of autonomic dysfunction in other body systems.

Extrinsic physical factors and intrinsic emotional stress are the commonest precipitators of autonomic dysfunction in the labyrinth.

Proper medical and psychiatric treatment can relieve and control most of the vasomotor disorders of the inner ear.

A small percentage require surgical destruction of labyrinthine components because of intractable autonomic dysfunction.

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INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY, LONDON, 1949.

The British Association of Otolaryngologists is organizing the Fourth International Congress of Otolaryngology, to be held in London from July 17 to July 23, 1949. There will be further meetings, for those who wish to go, at Oxford, Cambridge and Edinburgh on July 25 and 26. It is hoped that a full academic program will be arranged, and also various social functions.

The secretaries of the National Otolaryngological Societies have been circularized and asked to send a list of their members for individual notification. Should any association not receive this letter, they should communicate with the General Secretary, F. C. W. Capps, F.R.C.S., 45, Lincoln's Inn Fields, London, W.C. 2.

PRACTICAL ASPECTS OF LABYRINTHINE TESTS.*

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We live in a dizzy world. With each succeeding day, life becomes more complicated and living more complex. Tensions mount; conflicts increase in number and degree. The pace is too swift for some, and they fall by the wayside. This is literally as well as figuratively true. Some who drop out do so because they actually fall, victims of vertiginous attacks, whether fancied or real.

This would appear to be the reason why there is an increasing number of patients who say they are dizzy. Otolaryngologists do not see all who have this complaint. They get to examine only a small part of them. It is only when the interist or the general practitioner fails to effect a cure and the patient or the doctor is disturbed about the results obtained that members of the larger group are referred to the otolaryngologist. A recent patient, who had been examined and treated by two neurologists, because she had not obtained relief from her dizziness, came to me and complained bitterly that neither of the two had examined her ears. When examination revealed she had a labyrinthine disorder, she said, "I cannot understand this. How could they treat me when they knew neither nature nor degree of my disorder?"

Here in a nutshell is the problem. There is more dizziness, and there is some ineffectual examination of those who have it. It poses the question, "What can the otolaryngologist do about it?"

It is the purpose of this presentation to talk practically about labyrinthine examinations. One can easily become lost

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in a technical discussion of the physiology of the labyrinth and its central connections. When such experts as deKleyn, Versteegh, Dohlman, Hallpike, Kobrak, Speigel, McNally, Lindsay and others debate such subjects as nystagmus bereitschaft, utricular function, rhythmic phases of postrotatory nystagmus, postural reflexes, chronaxia and kindred problems, it is with difficulty that one follows their arguments. When one considers, too, that an authority like Kobrak says, "The psychic component of nystagmus is important. In testing for nystagmus, gross stimuli often produce considerable dizziness as well as nystagmus. When marked dizziness appears, increased intensity of nystagmus (of the postrotatory type) almost inevitably follows," and McNally states, "It is known that the labyrinthine reaction will vary within limits from day to day even when the method of applying the stimulus to the labyrinth is fairly constant," one wonders if there is any worth in labyrinthine tests, and if they should be used at all. This brings to mind the pleas of Lindsay and Walzl in their discussion of McNally's paper on Experimental Studies of Endolymphatic Flow, given before the Academy in 1947, and McNally's final summary, when each plead for simplicity of tests and urged that we should not try to deduce too much from too little. When we attempt to deduce a great deal from small variations, we can become fogged in our thinking and reach fanciful diagnoses which may have no sound basis at all.

These are reasons why a plea can be made for a practical approach to patients who have vertigo, and why in testing labyrinthine function practical tests should be employed. A description will be given of one of these.

Is it amiss to say that a good history is a practical labyrinthine test? I think not. Technically, it is not a test, but practically, its employment will eliminate many who claim dizziness as an outstanding symptom. Careful questioning will segregate most of the pyschogenics. Pinning a patient down to an accurate description of his ailment will sometimes be most informative. It is surprising how many patients will state they have dizziness, but in reality have only giddiness

or light-headedness on arising quickly from a sitting position. A few leading questions will classify others. The order of the appearance of symptoms will differentiate Ménière's from Lermoyez syndrome with consequent better prognosis in the latter. If good questioning brings out the fact that the patient is truly a victim of vertiginous attacks, some additional questions as to past illnesses, possible allergies, the function of the patient's gastrointestinal, cardiovascular-renal, endocrine and nervous systems, etc., may bring one almost as near to the etiology as subsequent examination will. This takes time. The majority of otolaryngologists build their office schedules around 15-minute appointments. No 15-minute appointment will adequately meet the demands of a truly dizzy patient. Sometimes the history alone will take more than that.

It is in the realm of physical examination that the otolaryngologist can score for himself and benefit the patient. If one is committed to the investigation of patients with dizziness, there must be a more complete examination than merely scanning the ears, nose and throat and pouring a little cold water in the ears. It takes but little additional time after a thorough examination of the ears, nose, nasopharynx and throat, to include a look at the pupils, a search of the eyegrounds, do a Romberg test, perform tests for spontaneous and positional nystagmus, to examine for gross neurological signs and order the necessary laboratory examinations. In most instances the responses to these tests will be normal, but before any caloric tests are attempted they should be performed. Let us stick to the practical. One does not need to be an ophthalmologist to examine the eyegrounds, but the gross lesions depicting intracranial pathology or advanced arteriosclerosis should be discernible to one who has looked at more than an occasional one. Nor do we need to be neurologists. It takes but a few moments to test a few reflexes, ascertain if ataxia is present and learn something about the sensory pathways. If these are grossly involved, we have diagnostic aids of worth. If more detailed and intricate testing is necessary to bring out the evidence of neurologic involvement, it is not the responsibility of the otolaryngologist to make the final diagnosis. This can

and should be the neurologist's. This keeps us in the realm of practicality.

Certainly, in all these cases, it is our responsibility to do careful hearing tests. Practically, they are of value. With vertigo of central origin it is unusual, except in the instance of acoustic neuromas and possibly certain cerebellopontine angle tumors, to have involvement of hearing. In vertigo of peripheral origin, if the hearing is good it is probable that the lesion in the labyrinth is not severe and the prognosis, therefore, is good. Marked hearing loss and vertigo is a combination which should put every otolaryngologist on his toes.

These matters attended to, there remains the functional examination of the labyrinth. What tests shall we use? Can we continue to be practical?

We are all inclined to continue to use the methods in which we were trained. Each of you is probably an advocate of the method you are presently using. My experience has been to change from one method to another. As I felt the shortcomings in one test, I found myself searching for a better. It was not until I had the privilege of seeing the work of Hallpike and Cawthorne, and learned their method, that I felt I had a reasonably satisfactory test of labyrinthine function. A description of this test and its suitability is the main purpose of this presentation.

Before the description of the test is given, it might be well to mention why, for me, some of the better known tests of labyrinthine function have seemed to be somewhat unsatisfactory. The turning chair tests both labyrinths at once, and while good information can be obtained from its use, there is always the disturbing necessity of differentiating the effect of one labyrinth upon the other. The results of mass douching with cold water, continued till nystagmus appears, vary according to the density of the medium through which the thermal stimuli (hot or cold) must pass before the nerve endings in the labyrinth are excited. The variation in time of the onset of nystagmus has never seemed to me to be truly diagnostic of labyrinthine function. An estimate of the ampli-

tude and rapidity of nystagmus of one side as compared to the other is certainly relative only unless there is a marked disparity. Minimal douching, according to the Kobrak method where 5 cc. of cooled water is used is controlled with difficulty. With the use of such a small amount of water, the time of injection into one ear, contrasted to the other, presents a variable which probably has some bearing on the end-result. The control of the temperature of the water is a hazard which is almost insurmountable. As a test to determine whether or not a labyrinth has function, this is probably the most convenient method of all, but for testing one side against the other, it has definite limitations. Douching with 1 cc. of ice cold water has some of the limitations just mentioned, and, while it easy for the examiner, it is not so for the subject. As a matter of fact, except for the Kobrak method, all of the above mentioned tests produce gross stimuli, which, it should be remembered, often produce marked dizziness and the latter gives rise to psychic reactions which may alter the degree and character of the nystagmic response. In the hyperirritable, and there are a good many of these, there results a feeling of nausea, pallor and sweating which is most uncomfortable. Occasionally there is vomiting. All of the information obtainable with these tests is available in the Hallpike-Cawthorne method, with the elimination of these undesirable side effects.

A description of the Hallpike-Cawthorne procedure follows. In the prone position with the head elevated at an angle of 30 degrees, the patient is tested with water at 7° above and 7° below body temperature; *i.e.*, 44° and 30°. Each ear is tested with both the hot and cold water. The testing fluid is regulated to the desired temperature, stored in an irrigating can placed some two or three feet above the head of the patient, and, with as little waiting period as possible, fed to the ear through a rubber hose and appropriate nozzle. The douching period in each instance is 40 seconds. Prior to each douching, the water in the tubing is run off and discarded. Marked variations in the temperature of the water in the tubing are posible. Hence this precaution. The duration time of the induced nystagmus is noted and charted in what

the authors have chosen to call a calorigram. During the period required for observation of the duration of the nystagmus the patient is instructed to look at a convenient mark on the ceiling directly overhead. The authors and others who have tried this method feel the use of Bartel's glasses or Frenzel lenses is contraindicated. This may prolong the reaction or diffuse the end-point. Observations, intermittent in the early stages of the reaction in order to avoid fatigue, are made from an advantageous position and the end-point of nystagmus noted. This is most easily accomplished by watching one of the small vessels of the sclera. Some practice is needed to determine with a fair degree of accuracy the moment of the cessation of the nystagmus. The recorded duration as noted on the calorigram extends from the application of the stimulus to the end of the nystagmic response. This eliminates the necessity of watching for and noting the onset of nystagmus. The charted duration on the calorigram is obviously not the duration of the nystagmus itself but the time interval from the start of the douching to the disappearance of the nystagmus. In other words, it is the nystagmus response to a standard stimulus. While attention is principally devoted to the duration time, the authors note that the degree of intensity of the nystagmus and the patient's subjective reactions (dizziness) should be recorded. A small difference in duration time between the two ears is of much more significance if it is associated with an obvious difference in intensity of nystagmus and dizziness.

The authors of this method note the results of tests in 50 individuals between the ages of 20 and 55 with normal hearing and without symptomatic or other evidence of vestibular disorder. In 80 per cent of these, the duration of the cold reactions varied between 1 minute, 30 seconds and 2 minutes, 10 seconds. In 80 per cent the cold reactions were in excess of the hot, usually by 5 to 15 seconds. In 10 per cent the cold and hot reactions were approximately equal, and in 10 per cent the hot reactions exceeded the cold with a maximum difference of 15 seconds. In 80 per cent the cold reactions of the two ears showed a difference of 5 seconds or less. In 10

per cent this difference was 10 seconds, and 20 to 25 seconds in the remaining 10 per cent. Differences of a similar order were observed in the hot reactions.

Fitzgerald, Hallpike and Cawthorne state that, "The test was evolved in a calculated endeavor to push to its limits the resolving power of the caloric tests," but they follow this by saying, "—this technique had greatly facilitated the recognition of abnormal reactions in patients with collateral evidence of vestibular disease." The former statement implies they are attempting to squeeze the last drop of blood from the life of the caloric test and that minimal differences between the two ears or between the hot and cold caloric stimulations in one ear are of prime importance. This is not true. They become practical when they say, in substance, that abnormalities of the test have more significance when these abnormalities are supported by collateral evidence of vestibular disease.

Criticisms of this test have appeared in the literature. Clarke, Glorig and Fowler, and McNally have pointed out that the 44° water reaches the ear at a temperature considerably lower than that figure and may be as low as 42°. They also note the temperature of the 30° water may not be that figure when it is delivered to the ear. It is argued from this that the test does not follow a standard and that the response to the hot stimulus cannot be compared to the cold as they are not equal stimuli. (Glorig and Fowler have used thermos jugs to obviate the temperature changes.) The point is academic. No one of these authors has noted the temperature of their patients at the time of testing. A variant here may be as significant as the changes noted above. The method should be considered from its practical aspects. It should be noted that the stimulus is as standard as any that has been devised and, most important, if all four douchings are carried out at one sitting the method tests the reactions of one ear against the other under these standards. One is not so much interested in the variation of nystagmus duration between the hot and cold stimulus in one ear as one is to learn the difference there may be between the two ears as they react to one or the other of the stimuli.

In addition to the fact that the stimuli to each ear are quite comparable, this method has advantages of worth. With the small temperature differences which the test employs, there is almost never an undesirable reaction. The douching is reasonably comfortable and the patient harbors no unpleasant memories of the test. Rarely does nausea come into the picture, nor the pallor and sweating of grosser tests. The method provides a check of reasonable worth. It is true that the duration responses to the hot and cold water are not usually exactly equal, but each checks the other, particularly when the four reactions are compared. (Incidentally, the reactions are repeatable. The duration response to the same stimulus in the same ear if retested will have no appreciable variation.)

Little will be said here of directional preponderance, a full discussion of which would change this presentation from the practical to the theoretical. In the great majority of subjects tested, the duration of nystagmus will be relatively equal for all four douchings, or the two douchings of one ear will be out of proportion to the two in the other. In directional preponderance the douchings which produce nystagmus in one direction (either to the right or left) are of longer duration than the douchings which produce nystagmus in the opposite direction. This finding has some clinical significance and is found in tumors of the temporal lobe, the direction of the preponderance being to the side of the lesion. It is also found in some instances of vestibular disease. The mechanism of directional preponderance is still under debate. Practically, it exists and often is confirmatory to other evidences of vestibular abnormality.

Place the findings obtained by this test alongside the results of the physical examination and the history, study the combination and the diagnostic evidence accumulated will be gratifying. Normal findings will sometimes appear when altered reactions are expected.

A very occasional bizarre reaction will ocur which will be difficult to interpret. In the main, however, it is surprising

how frequently one feels the test has been of material help in forming a diagnosis.

One illustration will perhaps suffice to show how the Hallpike-Cawthorne test is more informative than some other tests which are commonly used. Recently, an otolaryngologist referred a patient to our office. This patient had primarily a hearing problem, but there was in the history good evidence of attacks of dizziness. The referring otolaryngologist in an accompanying letter stated he had used 5 cc. of iced water in testing the patient's labyrinthine function. His conclusion was, that in addition to a hearing loss, this patient had evidence of slight hypoactivity of the right labyrinth. Without any intervening attacks of dizziness between our examination and the first, a Hallpike-Cawthorne test, showed no response to either the hot or cold stimulus in the right (involved) ear. Immediately following the test, cold tap water was douched into the right ear for one minute. Only then did a nystagmic response appear. The right labyrinth was not dead, but it certainly had more than a slight hypoactivity. When there is no response to the douching by hot or cold water according to the Hallpike-Cawthorne method, there is signal pathology in the vestubular apparatus.

Here, then, is a method of caloric testing which has a reasonable standardization of stimulus with an attribute (duration of nystagmus) which can be measured and which is often informative. In conjunction with allied examinations and a good history, its findings seem more conclusive than those elicited by other caloric tests.

Bilateral calorization and galvanic stimuli are adjuncts which may be used to obtain additional information to the above, but to the average otolaryngologist these are tests which belong to the finer realm of neuro-otology.

There is a great hue and cry heard these days about the diminishing scope of otolaryngology. Chemotherapy has robbed it of some of its most spectacular surgery. Surgeons in bordering fields are invading the precincts of the specialty.

The pathologies of the vestibular branch of the VIIIth nerve, including those of the end-organ and its central pathways, cover a field into which few have ventured; it has been left to the otologist. There is no reason why we whose medical interests include the diseases of the ear should not be as well equipped to examine the VIIIth cranial nerve as the ophthalmologist is the IInd. There is an increasing number of patients who complain of dizziness, and they are clamoring for better diagnosis and treatment. Clear thinking is required to separate the imagined from the real. Careful histories and examinations are needed to separate actual disease of the end-organ from conditions which alter the function of the end-organ. The same can be said for the differential diagnosis of lesions of the end-organ and central pathologies. Some suggestions of a practical approach to these problems and a practical method of examination have been presented. In this area there is an opportunity for better service than has been rendered in the past.

713 East Genesee Street.

MISSISSIPPI VALLEY MEDICAL SOCIETY MEETS AT ST. LOUIS, SEPT. 28, 29, 30, 1949.

The Fourteenth Annual Meeting of the Mississippi Valley Medical Society will be held at the Jefferson Hotel, St. Louis, Mo., Sept. 28, 29, 30, 1949, under the presidency of Dr. Alphonse McMahon, of St. Louis University. At the recent current meetings of the Society and board of directors, the following officers were elected: Dr. Nathaniel G. Alcock, Iowa City, Iowa, President-elect; Dr. Wendell G. Scott, St. Louis, Mo., First Vice-President; Dr. Charles F. Harmon, Springfield, Ill.; Second Vice-President; Dr. John I. Marker, Davenport, Iowa, Third Vice-President; Dr. Harold Swanberg, Quincy, Ill., Secretary-Treasurer; Dr. Ralph McReynolds, Quincy, Ill., Accounting Officer.

LABYRINTHINE SURGERY FOR MENIERE'S DISEASE.*†

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Ménière's disease is characterized by recurring attacks of vertigo, along with auditory symptoms of a distinctive type. The innear ear pathology which causes these disturbances is now known to be a hydrops of the labyrinth. The type of hydrops found in this condition differs essentially from that commonly found in inflammatory inner ear disease, but the etiology is indefinite and therefore it is called "idiopathic hydrops." The most favored theory as to the etiology seems to be that a disturbance in the secretary apparatus leads to oversection of endolymph.

The most predominating symptom of this disease and the one which causes greatest disability is the recurring vertigo. The most consistent symptom and the one which is necessary for diagnosis is the loss of hearing which begins as a low tone inner ear impairment. Other auditory symptoms which vary greatly but may be so severe as to be of great subjective concern are low-pitched tinnitus, a sense of blockage in the ear and a disagreeable disturbance of the quality of the tone, usually a diplacusis. The auditory symptoms fluctuate in severity and the hearing threshold varies widely from time to time, but the loss tends to become progressively greater.

The diagnosis is usually made when both auditory symptoms and vertigo are present. In the infrequent case, however, it is possible to make the diagnosis on the basis of the characteristic nature of the auditory symptoms without the presence of vertigo.

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The use of surgery in the treatment of the disease has been directed primarily at the prevention of vertigo. Although this has been consistently accomplished by a number of methods, no treatment can be considered wholly satisfactory unless the effect on the auditory apparatus is also favorable.

In view of the nature of the pathologic lesion in the ear, surgery cannot be expected to cause any great improvement in hearing. Also, since the disagreeable tinnitus and diplacusis probably take origin from the same cause as the hearing loss, it is unlikely that surgery can relieve them without destroying auditory function.

At the present time surgery is, therefore, used as a last resort in those cases which have not been sufficiently helped by medical therapy. The percentage in such cases varies according to different reports but would appear to be somewhere between 10 and 20.

Ménière's disease is a unilateral process in approximately 90 per cent. When bilateral the time of onset in the second ear varies. It may be soon or not until four or five years after the first. The hearing loss may be about equal or may continue to be greater in the originally involved ear.

In the bilateral case, therefore, a surgical procedure that may destroy hearing is unsuitable, except in the cases where the symptoms can be definitely ascribed to the ear with much the poorer function.

In the unilateral case the preservation of the remaining hearing is usually of little service to the patient except as a reserve that might be used in case of injury to or similar involvement of the other ear. This latter possibility, however, is of sufficient importance to warrant a search for a surgical procedure which might permit selective destruction of the vestibular function by the relatively safe intralabyrinthine route.

Intracranial division of the vestibular fibres of the VIIIth nerve, first performed by McKenzie,[1] and strongly recom-

mended by Dandy,[2] preserves auditory function. The method has given excellent results but falls short of an ideal treatment in that the process in the inner ear is probably not impeded or influenced, also that the intracranial approach may carry some operative risk if not expertly carried out.

LABYRINTHINE SURGERY.

Surgical procedures on the labyrinth have attracted much attention because of the simplicity with which they can be carried out and the absence of operative risk.

PORTMANN'S OPERATION.[3] DRAINAGE OF THE SACCUS. ENDOLYMPHATICUS.

Portmann's operation consisting of drainage of the saccus endolymphaticus was conceived before the pathological process was known. The discovery by Hallpike and Cairns[4] of a hydrops of the labyrinth in Ménière's disease appeared to strengthen Portmann's thesis that drainage of the saccus was a logical procedure. Portmann reported good results; but the experience of others in smaller numbers of cases has been less favorable, and the method is not in general use.

EXPERIMENTS ON THE SACCUS AND DUCTUS ENDOLYMPHATICUS.

In order to demonstrate the effect of simple drainage of the saccus and ductus endolymphaticus and also to obtain experimental evidence regarding the function of these structures in the maintenance of the volume of endolymph, a series of experiments was done in the monkey. These consisted of both simple drainage of the saccus and various degrees of destruction of the ductus endolymphaticus.[5] In three ears the saccus and medial convoluted part of the ductus, described as Sinus II by Anson and Wilson,[6] were completely destroyed. After a period of between three and one half and four months the defect was completely filled in by new bone and marrow, but the remainder of the endolymphatic system and the sensory organs showed no histological variation from the normal.

The conclusion seemed warranted from these experiments

that the maintenance of a normal volume of endolymph is not dependent upon a functioning ductus endolymphaticus or even upon its existence; also, that the occurrence of a hydrops or Ménière's disease could not be attributed to interference with normal absorption of endolymph from its dilated and convoluted middle portion.

It was also demonstrated clearly that drainage and partial destruction of the saccus was followed by sealing off and healing of the defect without any demonstrable effect on the labyrinth beyond.

Any effect of Portmann's operation on Ménière's disease would, therefore, be of only relatively brief duration.

ALCOHOL INJECTION.

The injection of alcohol into the labyrinth has been used chiefly in England. Mollison[7] opened the external semicircular canal and injected three minims of absolute alcohol after first withdrawing a similar amount of perilymph. Reports by him and also by Wright[8] and McDowall,[9] who used the same method, have been favorable as regards prevention of vertigo, although not completely successful. Deafness followed in all cases. Tinnitus has, however, remained undiminished in as many as one-third of the latter's cases.

Ablation of a Membranous Semicircular Canal: Removal of a part of the membranous horizontal semicircular canal has sometimes occurred accidentally during the fenestration procedure for otosclerosis, usually during a revision. Personal experience in two such cases was that a further loss of hearing occurred postoperatively, and the fistula response was negative. This was followed in several weeks by a return of hearing to the preoperative level for speech frequencies. The fistula response never became positive.

Although reports are lacking in the literature, personal communications indicate that the experience of others has been similar in at least some of their cases. There is, therefore, evidence that removal of part of the membranous canal

without its ampullary end destroys function of that canal but does not destroy hearing in at least many of the cases.

In the fenestrated monkey's ear destruction and obliteration of one canal produces no histologic change in the vestibule or in the auditory apparatus.

Cawthorne[10] has treated a large number of cases of Ménière's disease by removal of the ampullary end of the membranous horizontal canal apparently along with its ampulla.

He has reported consistently good results in preventing attacks of vertigo, but all cases were reported to be deaf in the operated ear.

It would seem that these results in Ménière's disease do not correspond to the experience with otosclerotics. Also, the histologic findings in the monkey's ear suggest that if the injury is limited to the canal and its ampulla, the auditory apparatus escapes injury.

Coagulation of the Labyrinth: Coagulation of the horizontal membranous canal, ampulla and the vestibule through a small fistula near the ampulla in cases of Ménière's disease was first reported by Day.[11] His results in preventing further attacks of vertigo have been uniformly good. He reported relief of tinnitus in nearly all cases and a preservation of useful hearing in approximately 10 per cent. He has had no complications from the operation. McNally[12] has reported one case, however, in which there was a facial palsy for several months following coagulation, thereby indicating that the facial nerve is not completely free from the danger of injury.

The experience of others, for example, Sullivan as reported by Ireland,[13] who have used the coagulation procedure in the vestibule, has been that profound deafness followed without exception.

The explanation of Day's more favorable experience suggests the possibility of some variation in technique, such as his stated practice of withdrawing the coagulating tip slightly

after it has impinged on the wall of the vestibule before applying the current.

The author's experience with the coagulation procedure in Ménière's disease has been limited to eight cases. In most cases the coagulation was limited to the membranous horizontal canal and its ampulla but was sufficiently strong and repeated so as to destroy these structures. In some the coagulation undoubtedly involved the utricle also. In one case the coagulation was applied to two canals at approximately a maximum distance from the ampullae. In all cases the hearing loss was increased to the point where no definite hearing could be demonstrated although tested over periods of years thereafter.

Prevention of attacks of vertigo was accomplished in five cases; one could not be followed up, and two cases developed occasional mild vertigo, which was apparently caused by the opposite ear.

In some cases there was intermittent low-pitched tinnitus after several months had elapsed and along with it a sense of blockage in the ear, which, although not so severe as before operation, was sufficient to cause some apprehension that it might be followed by an attack of vertigo.

Discussion: When the results of Day's cases are combined with those of others who have used the coagulation procedure and the experience with coagulation limited to the canals and ampullae only, it would appear that preservation of the hearing can be anticipated only occasionally or, at most, in a small minority of such cases.

The coagulation procedure has been successful as a means of preventing attacks of vertigo and subjectively has been a satisfactory treatment. In most cases the further loss of hearing causes no disability but it is of sufficient importance to limit this form of surgical therapy to a relatively small percentage of cases. An intralabyrinthine surgical procedure which would consistently preserve hearing while preventing vertigo would undoubtedly become a treatment of choice in a much larger group.

ANIMAL EXPERIMENTS ON COAGULATION AND INTRALABYRINTHINE NERVE INJURIES.

A series of experiments on the rabbit's ear done in our laboratory by Dr. Bruce Proctor before penicillin was available, but not reported, showed that instrumentation within the vestibule in the attempt to rupture or injure the utricle resulted in extensive fibrosis in the perilymphatic spaces and varying degrees of hydrops and degeneration of sensory organs. These results, along with early experiences with operations on the human, led to further experiments to determine the extent to which the vestibular apparatus might be injured by coagulation or instrumentation without causing histologic changes in the auditory sense organ. More recently the effects of the use of the coagulating current within the vestibule have been demonstrated by Schall and Rambo.[15] These have confirmed the improbability of preserving hearing in more than a minority of cases when treated by coagulation within the vestibule.

The following experiments were done on the monkey's ear during the past several years to determine their effect on the histologic appearance of the auditory sense organ. Coagulation of the ampulla of the horizontal canal. Coagulation of all canals at maximum distance from the vestibule. Injury to the nerves to the horizontal canal ampulla and to the utricle, and in one case detachment of the otolithic membrane of the utricle. Introduction of the coagulating tip and the use of current which was not strong enough to cause bubbling or visible changes, although sufficiently strong to produce a facial nerve stimulus.

All ears were prepared and examined histologically. In practically all cases histologic evidence of the injury to the vestibular apparatus was demonstrable. It was found that in no case had the injury resulted in any extensive change or fibrosis in the perilymphatic space of the vestibule.

In one animal sacrificed within 24 hours of coagulation there were collections of red blood cells in the perilymphatic spaces and an altered staining reaction of the perilymph, but

in all others sacrificed after several days or weeks there was no evidence of hemorrhage.

None of the procedures involving limited instrumental injury to the ampullary nerve and utricle or coagulation which was limited to the canals and ampullae resulted in any histologic change in the auditory apparatus. In no case was there more than a minimal amount of fibrous tissue in the perilymphatic cistern.

In the ear which was fixed within 24 hours after the coagulation there was some suggestion of a chemical change in the perilymph, as indicated by the altered staining reaction.

The histologic findings in these ears failed to provide an explanation for the profound loss of hearing which occurred in humans in which the coagulation procedure had been limited to canals or their ampullae only.

SIMPLE FENESTRATION, DECOMPRESSION OF PERILYMPH, INTRALABYRINTHINE NERVE INJURIES.

In the search for a method to prevent vertigo while preserving cochlear function, several procedures were carried out on humans with intractable Ménière's disease.

Case 1: G. R., male, age 40, advanced unilateral Ménière's disease unrelieved by medical therapy. Serious disability from vertigo and average hearing loss of over 60 db. in speech frequencies. This patient had a previously healed radical mastoid cavity. On July 15, 1940, a fenestra was made near the horizontal canal ampulla and a barbed dental broach passed through the ampullary nerve into the vestibule in an anterior direction until it impinged on the opposite wall and then was withdrawn. This was repeated. No bleeding or visible injury to the membranous canal or its ampulla was observed despite the use of the barbed instrument. The fenestra was covered with a skin flap. The hearing in the ear was further impaired to a state of profound deafness. Vertigo was relieved except for mild recurrences on infrequent occasions, but no longer caused any disability.

Although the ear was profoundly deaf, tinnitus and a sense of blockage of mild degree have been intermittenly present. The fistula response has remained active and the fenestra is visible through the thin covering skin. The response to cold caloric stimulation with ice water was absent two months following operation, but has become active to an approximately normal degree since that time. There has been no Tullio phenomenon.

Case 2: E. B., female, age 32. Unilateral Ménière's disease for three years, unrelieved by medical therapy. Fluctuating hearing threshold averaging about 40 db. loss for speech frequencies. On April 3, 1945, a simple mastoidectomy was done and a fenestra made in the horizontal canal near

the ampulla. An unbarbed dental broach was passed anteriorly through the nerve to the ampulla until it impinged on the wall of the vestibule in the attempt to damage the nerve to the utricle as well. This was repeated. There was no visible injury to the membranous canal or ampulla and no hemorrhage.

The middle ear conducting apparatus was avoided entirely. No covering was applied to the fenestra. A few sulfathiazole crystals were sprinkled in the cavity and the wound closed. Penicillin was given for several days thereafter.

The wound healed by first intention. Afterwards, deafness was found to be profound in the operated ear. The fistula response remained strongly active, on pressure, strenuous nose blowing and sneezing. A Tullio[14] reaction was present on exposure to loud sounds or noises, and caused great annoyance. Although the ear was deaf, an intermittent sense of blockage and tinnitus presisted. While the fistula response was strongly active, the cold caloric response was found to be diminished with the head at 60 degrees backwards. Attacks of vertigo were infrequent but of sufficient severity that a secondary operation was done on June 12, 1946, over 14 months after the first operation. At this time the mastoid cavity was found to consist of one large air cell, the lining mucoperiosteum of which formed the covering of the fenestra, which was widely open.

The membranous canal and ampulla were then destroyed by coagulation, the coagulating tip extending slightly into the vestibule.

After operation, vertigo was moderate, receding gradually over several weeks. No further attacks of vertigo had occurred when last observed several months after operation.

Case 3: E. M. H., male, age 55. Unilateral Ménière's disease for two years, which had resisted medical therapy and was a serious hazard for his occupation of railroad switchman. There was an average hearing threshold loss of about 35 db. for speech frequencies.

On May 12, 1945, a simple mastoidectomy was done and a fenestra made as in Case 2, with a similar attempt to injure the nerves to the horizontal canal ampulla and utricle. The wound was closed in a similar way, with no covering being placed over the fistula. Sulfathiazole crystals were dusted into the wound and penicillin therapy given parenterally postoperatively. Recovery was rapid. The ear was profoundly deaf after operation and has remained so until the present time. There is still slight hissing type of tinnitus in the ear, but the severe noises have been relieved. An active Tullo[14] phenomenon has been present since operation and is activated by such sudden sounds as a sudden escape of steam from a locomotive, the grinding of car wheels and ringing of the engine's bell close by.

The fistula response remains strongly positive on pressure over the mastoid scar. There have been occasional mild attacks of dizziness, but patient considers himself sufficiently relieved to remove the hazard and he wishes no further treatment.

Case 4: J. B., male, age 51. Ménière's disease in the right ear of about 20 years' duration, with threshold hearing loss of over 60 db. for speech frequencies. He also had a threshold hearing loss in the left ear which was less marked, but which showed wide fluctuations for speech frequencies. Although patient referred most of his complaints to the more impaired ear, a diagnosis of early Ménière's disease in the better ear seemed indicated as well.

For the purpose of obtaining further information, a two-stage operation on the more impaired ear was decided upon. On Oct. 11, 1946, a simple

mastoidectomy was done as in Cases 2 and 3. There was no mastoid pneumatization, but a moderately large antrum was found. A simple fenestra was made without injuring the membraneous labyrinth and without attempting any interference within the bony labyrinth. No covering was placed over the fenestra. The wound was closed after dusting in a few sulfathiazole crystals and penicillin was given for several days.

Recovery from the operation was rapid. The ear which had about 60 db. threshold loss for speech frequencies before operation was totally deaf to a loud shout and to the audiometer thereafter when the opposite ear was masked.

An active fistula response persisted, as well as annoying vertigo whenever he was close to loud noises (Tullio phenomenon). The tinnitus was not relieved and attacks of vertigo persisted, as well as that which occurred because of the open fistula. Seven months after the first operation the wound was reopened for inspection of the fenestra and coagulation of the membranous labyrinth. The antrum was again pneumatized and the fenestra found to be widely open and covered by the semitransparent mucoperiosteum. The canal and ampulla were then coagulated, the coagulating tip being extended into the vestibule but not to the opposite wall.

Following coagulation, there has been relief from the Tullio reaction and from the vertigo related to the open fenestra, but there have also been occasional spontaneous attacks of vertigo which may have originated from the other ear.

Tinnitus has also been present, but not well localized to either ear.

DISCUSSION.

The experience with these cases has permitted several observations of significance:

1. Decompression of perilymph is of no value as a relief to Ménière's disease, or hydrops of the labyrinth.

2. A fistula made in a bony semicircular canal through a simple mastoid approach and left uncovered will probably remain a permanently open fenestra.[14] This is undesirable because of the associated fistula symptoms and Tullio phenomenon. Also, a fenestra covered only by mucosa might offer less resistance to infection in case of subsequent middle ear suppuration.

Closure of a fistula can be assured at operation by making a very small opening and leaving bone chips in it, and probably also by destruction or removal of the membranous labyrinth.

3. The depression of the hearing threshold to a state of profound deafness in all four of these ears, including that in

which only a simple festra was created, suggests that the existence of a third window into the labyrinth in the presence of an intact conducting mechanism results in a definite threshold loss.

Whether such a hearing loss in the first three cases would have been avoided by causing the fistulas to close, thereby limiting the procedure to the intralabyrinthine injury, has not yet been determined.

SUMMARY.

1. The prevention of attacks of vertigo, along with destruction of auditory function, has been formerly accomplished by labyrinthectomy operations, all of which involve extensive destruction of structures in the vestibule. Efforts to prevent vertigo by intralabyrinthine surgery without causing destruction of hearing have so far met with limited success. The following procedures have been done on humans:

a. Portmann's operation on the saccus endolymphaticus. This operation has preserved the hearing but has failed to give sufficiently good results in prevention of vertigo.

Experiments on the monkey have shown that drainage or destruction of the saccus, as well as nearly all of the ductus endolymphaticus, is followed by prompt healing and, therefore, could have only a limited effect on a hydrops of the labyrinth.

b. Ablation of the ampullary end of the horizontal semicircular canal has been reported by Cawthorne to prevent attacks of vertigo consistently, but also to cause deafness.

Accidental removal of part of the membranous canal at the fenestration operation or its revision causes temporary deafness, but hearing frequently returns to the previous level.

Obliteration of the horizontal canal for several millimeters distal to the ampulla in the monkey causes no histologic change in the auditory apparatus in most instances.

c. Coagulation of the ampullated end of the horizontal canal,

with the tip of the instrument extended into the vestibule (Day's procedure) has prevented vertigo attacks consistently. Preservation of hearing occurred in about 10 per cent of Day's cases but not in those reported by others.

d. Coagulation limited to one or more canals without entering the vestibule has prevented attacks of vertigo but caused profound deafness. It did not always abolish intermittent tinnitus and sense of blockage in the ear.

In the monkey, it caused no histologic evidence of injury to the auditory apparatus.

e. Decompression of perilymph is of no value in treatment of Ménière's disease.

f. A simple fistula in the horizontal canal, if made after simple mastoidectomy and left uncovered, the mastoid wound being closed, remains as an open fenestra beneath the mucosa which later lines the mastoid antrum. Such a fenestra in the presence of Ménière's disease is undesirable because of the further loss of hearing which occurs, the disturbing Tullio reaction and the possible tendency to facilitate infection of the labyrinth in case of a middle ear infection.

Closure of a fistula can be assured by keeping it a minimum size, leaving bone dust in it, and by destroying the membranous canal.

g. The histologic evidence obtained from monkey experiments as well as the experience of Day in his use of coagulation on the human lend encouragement to the search for a means of interrupting vestibular function without injurying the auditory apparatus. Further investigations are being carried out in which sound conditioned animals are utilized as well as histologic controls.

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THE ROLE OF VIRUS INFECTIONS IN THE ETIOLOGY OF SEROUS OTITIS MEDIA.

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The increasing incidence of secretory otitis media in the last few years has led to a renewed interest in the etiology, diagnosis and treatment of this disease. The diagnosis, which it would seem should be simple and self-evident, is frequently difficult and often escapes the attention of the examiner.

Hoople,[1] in relating his experience in otolaryngology during the late war, chose this subject for discussion before the Royal Society of Medicine in London and stated that, in his opinion, it was the most frequently missed diagnosis in otology. Eagle,[2] on noting a sudden increase in the disease, beginning about 1941, states "we are reluctant to admit that the disease had been prevalent in the years prior to 1941 and that our eyes are now seeing pathology which they had previously failed to see." Hantman,[3] in an excellent article in 1943, called to the attention of the Army Medical Corps the frequency of the disorder and the importance of the correct diagnosis.

Since secretory otitis media has been for a long time a well recognized clinical entity, why should there have been a sudden increase in its incidence and an apparent difficulty in its recognition? The latter question may be answered by the fact that, although the disease has long been recognized, its incidence has been comparatively rare and usually recognized only when a fluid line could be distinctly seen through a transparent membrane. To account for the unquestionable increase in the incidence of the affection, we must look for causes other than those which have been recognized in the past.

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The commonly accepted etiology of serous otitis media is essentially mechanical — namely, an obstruction of the Eustachian tube due to a number of causes, such as the presence of adenoid tissue, or a tumor mass in the nasopharynx, infection of the nose, pharynx and sinuses, external pressure on the tube by swollen lymph nodes and, as pointed out by Robinson,[4] by passive congestion secondary to pressure on the pterygoid plexus.

Aerotitis must be recognized as the outstanding example of the mechanics of the blocked tube, but the sequence of events is so rapid and the pressure changes so great in degree as to be in no way comparable to the much slower progression of an acute serous otitis media.

It would seem questionable if obstruction of the tube, without any inflammation or irritation, could alone be a common cause of effusion into the middle ear. The dry tube obstructed by a stricture, necessitating the use of forcible bouginage in order to open it, is very rarely the cause of such an effusion. Even the classic example of effusion due to obstruction of the tube by malignant disease may be questioned — as witness, one patient suffering from this disease in whom the tube could be easily inflated. In this instance the effusion must have been influenced by irritation from the advancing malignancy or interference with lymphatic drainage.

Politzer[5] says, "Exudation into the tympanic cavity is most frequently caused by the inflammatory process of the mucous membrane itself; nevertheless, it is beyond doubt that sometimes in excess swelling of the tubal mucous membrane and impermeability of the Eustachian tube there occurs, in consequence of the consecutive rarefaction of the air in the tympanum, a transudation of serous fluid which is free of bacteria."

The common cold in the head, certainly the most frequent combination of infection, plus partial closure of the tube, has not in the past been a prolific cause of the effusion and does not account for the recent increase in the disease.

In a search for other causes, two immediately come to mind: first, avirulent infections which have long been considered as a contributory factor; second, new types of infections. The possibility of allergy as a cause of the effusion has been kept in mind constantly and in no instance has it proved to be an etiologic factor.

In regard to avirulent infections, there seems to have been a distinct decrease in the virulence and morbidity of acute otitis media during the last decade, analagous to that of scarlet fever which, likewise, has declined in virulence and morbidity and for no apparent cause.

In reviewing the history of acute and subacute otitis media, there immediately comes to mind a picture of the virulent overwhelming infections of the middle ear and mastoid, with the constant threat of septicemia and meningitis which characterized the first quarter of this century. The sudden onset with high fever, severe pain, early rupture of the drum, mastoid tenderness and postaural edema appearing in from three to five days from the onset, and the drooping of the canal wall, all conformed to the typical textbook picture which is so seldom seen today. The streptococcus and pneumococcus were the prevailing organisms. Shortly after World War I, a more insidious infection began to appear, which was due to the streptococcus mucosus capsulatus, now known to be the pneumococcocus type III. This infection was characterized by a slow, insidious onset with gradual involvement of the mastoid from the antrum outward to the cortex, with mastoid tenderness and edema only becoming evident as late as the second to fourth week. Frequently the duration of the infection and repeated X-ray examinations were the only indications of the progress of the disease.

During the 'thirties, coincident with the subsidence of the virulence of these infections, there began to appear more conservatism in performing paracenteses. At the Children's Hospital in Boston a rule was put into effect that no paracentesis should be performed without examination and consent by the department of otology — whereas, it had previously

been the custom for the medical resident to perform the operation on any drum membrane which appeared to be inflamed. The result was an immediate decrease in the incidence of suppurative otitis media and mastoiditis. At about this time, also, the necessity for ligating the jugular vein was being questioned more often.

We then arrive at the period ushered in by the introduction of chemotherapy in about 1936. Although experimental work had been carried on for several years before 1936, it was not until 1938 that the use of these drugs became at all general. The first drugs proved to be somewhat toxic and it was advisable to hospitalize all patients for careful observation during its administration. New preparations were rapidly introduced, each one less toxic than its predecessor. In order, they were prontosil, sulfanilamide, sulfapyridine, sulfathiazole and sulfadiazine.

During 1941 there was a marked decrease in the number of simple mastoidectomies performed. At the Massachusetts Eye and Ear Infirmary the number dropped to 36 per cent below the previous 10-year average, at the private ward of the Childrens' Hospital to 56 per cent below the 10-year average. At the former institution this loss has held and has even increased up to the present time. At first glance, one could give the entire credit for this decrease to the new drugs, but it must be remembered that at that period we were feeling our way in their use in constant fear of their toxicity and they were not prescribed as freely as is now possible with penicillin. Warnings of the masking effects of the drugs were also frequent.

Many have suspected that we have been passing through a period of avirulent infections. Butler,[6] in the late 'thirties, had occasion to compare the results of treatments, with and without chemotherapy, in two small series of acute suppurative otitis media and found the incidence of operative mastoiditis to be the same in each series; therefore, as chief of otolaryngology in a large station hospital from September, 1942, to March, 1944, he instituted a nonchemotherapeutic regime for

the treatment of acute middle ear infections — needless to say with consent from higher authority. He reports: "In this series of 610 cases of myringitis, exudative otitis media and purulent otitis media, treated without sulfonamides or penicillin, only two surgical mastoidectomies were necessary. If only the 461 cases of purulent otitis media are considered in regard to the two mastoidectomies, it would seem that the morbidity of ear infections had paralleled the known decrease in the morbidity of such conditions as measles and scarlet fever."

It must be understood that in this argument to establish the probability of a period of avirulent infections during the last few years, it is not in any way meant to minimize the importance and advantages of chemotherapeutic agents and antibiotics in combating infection.

If, then, we may assume that we have been experiencing a period of avirulent infections, perhaps a cyclic change such as has been noted in previous decades, the increased incidence of serous otitis media can be explained, and especially so if serous, exudative and suppurative otitis media are considered as sequential stages of the same process. It is only fair to state, however, that at the present moment there are indications of an increasing virulence of these infections.

As to the possibility of new types of infections: It has been interesting through the years to observe the protean manifestations of the "grippe" or influenza epidemics, each of the same general pattern but exhibiting some bizarre symptom such as tenderness of the scalp, neuralgia of the face, frontal or occipital headache and various myalgias peculiar to the particular season.

Of late years, however, there has developed a type of upper respiratory disease which is typical of neither influenza nor of the common cold, and is characterized by the prominence of pharyngitis and tracheitis, with a relatively insignificant coryza. During the winters of 1945-1946 and 1946-1947, many patients presented themselves, complaining bitterly of dysphagia of four to five days' duration, yet on examination the

pharynx appeared to be perfectly normal. This period proved to be a sort of prodromal stage to a very evident pharyngitis and tracheitis with severe cough, while nasal and nasopharyngeal involvement was very mild. Malaise has been a prominent symptom — the temperature normal or only very slightly elevated — leucocyte counts normal or subnormal. The course of the disease has been prolonged and erratic, with frequent and irregular exacerbations, and the prolonged cough has suggested the possibility of a primary atypical pneumonia. Secretory otitis media has been a frequent accompaniment of this infection and, indeed, three instances have been noted in which the ear symptoms have preceded the onset of the pharyngitis and tracheitis.

Recent investigations have defined a new type of infection with symptoms similar to those just described. Enders,[7] in a "Review of Some Recently Defined Virus Diseases," before the Massachusetts Medical Society in May, 1947, had this to say: "During the decade 1920-1930 when I first began the study of infectious agents, it was more or less generally agreed that the era of etiologic discovery, if not definitely ended, was at least in its final stage. The efforts of bacteriologists in the future were to be directed to the more intensive study of the known pathogenic organisms — the number of which would not be materially increased — with a view to defining more precisely their metabolism, their chemistry, the immune reactions that they elicited and the means by which their attack might be prevented or withstood; however, we failed to visualize that rapid extension of its boundaries that was so soon to come. Since 1930, not only have the causative agents of such old and important diseases as yellow fever, influenza and mumps been determined, but also the etiology of a large group of other infectious maladies, many of which had been clinically unrecognized, has been established."

He then gives a list of seven diseases in man for which, within the period from 1940 to 1946, either conclusive or presumptive evidence of viral etiology has been presented. Among these is primary atypical pneumonia.

"After this list was prepared, a new virus disease was reported by a group of workers in Tennessee. An outbreak of epidemic meningoencephalitis accompanied by vesicular pharyngitis affected over 200 persons, and inoculation of filtrates of nasopharyngeal washings from certain patients into the embryonated egg resulted in the isolation of what is tentatively described as a virus differing from any of the known agents."

The etiology of primary atypical pneumonia had presented a baffling problem to investigators ever since this condition had been recognized as a clinical entity in the years just preceding the war.

"Accordingly, the Commission on Respiratory Diseases, located during the war at Fort Bragg, N. C., undertook experiments in human volunteers with the objective of proving whether the agent was transmissible and filterable. The results of these elaborate and costy experiments leave no reasonable doubt, I believe, that atypical pneumonia in which none of the known bacterial, fungal, viral or rickettsial agents can be demonstrated is due to a filterable virus.

"The Commission also concerned itself with the relation between two other frequent clinical types of acute respiratory illness and their etiologies. Thus, in additional experiments with 42 vounteers, they inoculated material obtained from a person with symptoms mainly of coryza — that is, those of the common cold and with minimal signs of pharyngeal inflammation. Another group was inoculated with material from a patient with an infection distinguished from the common cold principally by sore throat and little nasal involvement. The results indicated clearly that both these clinical types of infection could be experimentally transmitted to human beings by means of filtrates of throat washings. The most interesting finding, perhaps, was the difference in the incubation period of the two types of experimentally induced diseases. The coryzal syndrome had an incubation period of one or two days — an observation in agreement with earlier work on the common cold by Dochez and others. In con-

trast, the disease characterized chiefly by inflammation of the pharynx had an incubation period of five or six days. It will be noted that both these periods of incubation were shorter than that of the experimental atypical pneumonia. Worthy of emphasis, too, is the finding that no immunity apparently resulted from the experimental coryzal infection, since reinoculation within three weeks with active material from patients who had recovered again induced the disease. On the other hand, the pharyngitis virus produced a specifically increased resistance following reinoculation. Neither the common cold nor the pharyngitic syndrome induced immunity against experimental inoculation of material containing the virus of atypical pneumonia. The participation of hemolytic streptococci or influenza virus in either type of infection was satisfactorily eliminated as a possibility."

It is interesting to note that the pharyngeal type of infection with an incubation period of five to six days corresponds to the type previously described which is characterized by several days of severe dysphagia with negative findings and was considered to be the prodromal stage of an upper respiratory infection frequently foreboding a secretory otitis media.

The majority of these patients are seen during the period of effusion with its characteristic symptoms, but fortunately there have been numerous opportunities of observing the process from the very beginning and the sequence of events resolves itself into three stages:

1. A short period of mild pain or aching, accompanied by a sense of fullness in the ear.

2. An increasing hyperemia of the mucous membrane of the middle ear.

3. The period of effusion.

1. If the patient is examined a short time after the onset of the mild pain and sense of fullness in the ear, there will be observed an injection confined to the region of Shrapnell's membrane and at times extending to the upper posterior quadrant — rarely, if ever, does it extend beyond this area; if so,

we are probably dealing with an exudative type of infection. The rest of the membrane is essentially normal, retaining its luster and the light reflex remaining normal for that particular membrane. The pain is never as severe as that encountered in the "influenzal ear" or acute myringitis and there is no tendency toward the formation of blebs. The hearing is essentially normal and the tube inflates readily. At this point the process may tend to resolve, if not it progresses to the second stage of increasing hyperemia of the mucous membrane of the midle ear. The injection of the drum membrane, meanwhile, shows no tendency to spread and may, indeed, decrease in extent. At this stage, the process is also still capable of resolution, but more frequently goes on to stage III — of effusion.

It will be observed that the above discription is applicable only in the presence of a normal drum membrane through which the fluid line can be seen. It may be, perhaps, that some of the difficulty in diagnosis occurs in the presence of a membrane which is opaque or abnormally scarred and retracted and through which it is impossible to detect the presence of the fluid. It is well, therefore, to keep in mind the symptoms which are typical of the affection. These are a persistent sense of fullness and deafness in the ear, which at times may be relieved by positional changes of the head, an occasional "crackling" sound which is caused by air entering the middle ear and bubbling up through the fluid. Not infrequently the patient makes his own diagnosis when he complains of a sensation as of "water in the ear," such as is experienced after swimming; also, a frequent complaint is of a sensation of movement in the ear, as of a bubble in motion. The diagnosis may be aided by inflation when a sound as of air bubbling through fluid may be auscultated, but this seems only to occur when the fluid covers the opening of the tube into the middle ear, and if this sign is depended upon and fails, the presence of the fluid may be overlooked.

The history also is of extreme importance. Frequently the discomfort experienced from a small amount of fluid in the ear is so slight that many weeks and even months may have

passed before relief is sought; careful questioning, however, will elicit a history of an acute respiratory infection which also may have been so mild and evanescent as to have escaped the memory of the patient.

Given a history of infection plus the symptoms as described above, which do not clear up promptly under proper treatment, then an exploratory puncture, followed by suction with the Siegel otoscope, will often result in the evacuation of the fluid, much to the satisfaction of the operator and relief to the patient. Such a sequence of events up to but not including the paracentesis could well be assumed to constitute a symptom complex which is indicative of a serous otitis media.

It is hoped that enough evidence has been adduced to indicate that acute and subacute serous otitis media, in the majority of cases, is caused by a specific avirulent infection of the mucous membrane of the middle ear, originating perhaps in the upper respiratory tract and extending into the middle ear through the Eustachian tube or by a new type of infection, a virus peculiar to the pharynx and which, at times, seems to be primary in the middle ear itself, and that obstruction of the much maligned Eustachian tube is of secondary importance.

This concept is important in regard to treatment which should be directed, in the first instance, to the upper respiratory infection and at the same time with frequent inflations of the ear by the Politzer method; rarely is it necessary to resort to the catheter. The danger of introducing suppurative infection into the middle ear in this affection by inflation was disproved by both Politzer and Bezold years ago. During the third stage of effusion, treatment should be directed primarily to the middle ear, but any remaining infection of the pharynx should not be neglected. If the amount of fluid is small, it may yet be absorbed or, theoretically, be drained through the tube into the pharynx, although the latter possibility is doubtful, and treatment directed to the tube alone will be found to be ineffective. The most effective procedure is to evacuate the fluid from the tympanum by aspiration with a syringe, or

what will be found to be more effective, puncture of the membrane, followed by suction with the Siegel otoscope and Politzerization. This pinpoint puncture is sealed over within a few hours, but if not, and drainage continues for several days, the presence of a low grade suppurative infection should be strongly suspected.

It will be found that, as a rule, the less the amount of fluid the more quickly is the condition healed and that one treatment as outlined will frequently suffice; in others, several treatments are necessary. In a few instances, however, the fluid persists for an indefinite period in spite of repeated evacuations, which circumstances leads to a consideration of the involvement of the mucous membrane of the mastoid process.

In 1922, Jervey[8] reported a case of "mastoiditis hyperplastica Serosa," in which he queried, "If serous labyrinthitis, why not serous mastoiditis?" Lawson,[9] in 1927, also reported a case of "serous otitis media" with involvement of the mastoid, both cases being cured following a simple mastoidectomy. Lowy,[10] as quoted by Hantman, states that the cells of the mastoid process are practically always filled with exudate and on X-ray there may be shading of the whole pneumatic system. Cody[11] similarly, in 1941, reported four instances of involvement of the mastoid which were relieved by operation. A further report on several cases may help to emphasize some of the salient features of the disease.

H. D. S., male, age 58 years, was first seen on Oct. 13, 1939, complaining of a blocking and sensation of motion in the ear for several weeks following a cold in the head. On examination all signs of the upper respiratory infection were absent, the membrana tympani was within normal limits and no evidence of fluid could be seen. The middle ear was inflated readily without signs indicative of fluid and the relief was immediate. Four days later, he reported with the same symptoms, when, following inflation, a fluid line appeared at the level of the umbo and could be seen to ascend and disappear into the region of the aditus. The membrane was then punctured and considerable serous fluid evacuated. This sequence of events occurred at two-day intervals for two weeks, at which time all objective signs of fluid had disappeared, although he continued to complain bitterly of the sense of blocking and fullness over the entire side of the head, which was relieved only momentarily by the inflation.

Two weeks later, or four weeks after the first examination, the fluid was again evident, showing the same characteristic location in the upper

posterior quadrant and on evacuation seemed to be in greater quantity than could be accounted for by the capacity of the middle ear cavity. X-ray examination at this time disclosed a definite clouding of the entire mastoid without any evidence of destruction. The status remained the same for the next four weeks, with recurrent evacuation of the fluid, a series of three X-ray treatments were of no benefit, the fluid remained serous in character, cultures were negative, the temperature remained normal, no leucocytosis, and there was no mastoid tenderness. Operation was advised and accepted. At operation the cortex was found to be thin and extremely hard, the cell walls were hard and brittle, except in the region immediately surrounding the antrum, where they were slightly softened, the mucous membrane lining throughout was edematous and slightly hemorrhagic, especially in the cells extending from the antrum to the tip of the mastoid and in the posterior tip cells, while that of the antrum itself and the attic remained thin, white and perfectly normal in every respect. Recovery was rapid and without incident. The cultures were negative and the pathological report was serous mastoiditis without evidence of malignancy. There has been no recurrence of the disease and the hearing remains essentially normal for his age, at a level of 5 db below the unaffected ear.

F. D. A., male, age 53 years, was subjected to annual infections of both maxillary sinuses for years. In 1925, an intranasal antrotomy had been performed, and in 1926 an infected cyst removed from the right antrum by the Caldwell-Luc approach; nevertheless, the yearly attacks continued as usual. In February, 1937, and May, 1942, he suffered his first attacks of serous otitis media, both of which responded promptly to treatment. In August, 1943, following an air flight, he again had a serous effusion into the right antrum which resisted all treatment up to March, 1944. These treatments consisted mainly of inflations, several paracenteses, one course of sulfonamides and one of penicillin, as well as two with diathermy, the second of which was followed by a severe vertigo of one hour's duration. From January to March, 1945, the fluid was constantly present, but healed spontaneously. During June and July, 1945, he received three radium treatments to the nasopharynx during a period of quiescence. During December, 1945, in conjunction with an acute sinusitis, the fluid recurred and again resisted all treatments. X-ray revealed an increased density of the mastoid, with no evidence of destruction or decalcification. At operation, the cortex was found to be extremely hard, the cell walls were not decalcified, and the mucous membrane very edematous, except in the region of the antrum.

In both instances the middle ear seemed to act as a receptacle to receive the overflow from the affected mastoid cells. The pathology was reported as a serous mastoiditis and the bacteriological report was negative.

Mrs. A. L., age 24 years, was first seen on April 1, 1947, complaining of a "dull throbbing" and full sensation in the left ear, which had been present for nearly nine weeks following the use of the telephone, although at that time she was not conscious of any acoustic trauma, and there was no history of an acute cold. Except for a slight injection of the handle of the malleus, the examination at that time was essentially negative; there was no evidence of a subacute respiratory infection, the drum membrane was within normal limits, and the tube was patent. The hearing was essentially normal, a whisper being heard at 15 feet. Two weeks later, she again reported with the same symptoms, but somewhat more pronounced. At this time, the membrane appeared flat, grayish and thick, and puncture of the membrane, followed by Politzerization through

an easily inflatable tube, evacuated a few drops of clear, thick mucus. Three days later, there was still a slight discharge, a fact of some significance, in that in the purely serous affection the puncture wound tends to close in a few hours. Penicillin treatment was begun then, with 300,000 units in wax on two successive days, followed by 200,000 units by mouth every four hours for one week, with what appeared at first to be some improvement but which proved to be only temporary, although the discharge ceased. Another paracentesis disclosed a thick, cloudy mucoid discharge, the culture from which was reported as staphylococcus albus. The X-ray revealed a diffusely clouded mastoid without evidence of destruction in any way comparable to the serous type, but operation disclosed a broken down mastoid process with granulations on both the sinus and the dura. A large amount of thick mucoid material was removed from the attic and middle ear by suction. The culture from the mastoid proved to be sterile, probably due to the action of the penicillin.

This case is cited to illustrate the similarity in the time element and symptoms of the two affections, the difference being in the appearance of the drum membrane and the character of the discharge.

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MACROCHEILIA.*

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Macrocheilia may be defined as an abnormal or excessive enlargement of the lip and is not a common occurrence. Wood[1] states that certain of the enlargements of the tongue and lip known as macroglossia and macrocheilia are undoubtedly lymphangiomatous in nature. Ormsby[2] refers to macrocheilia as due to simple lymphangioma of the lip. Bloodgood[3] remarks on the many examples of exaggerated appearance of the labial glands throughout the mouth. Case reports in the literature supply meager accounts of the clinical and pathological data pertaining to the condition, so that an accurate diagnosis of the underlying etiology and pathology is rarely possible. New and Kirch[4] state that the etiology is not known. Conway's[5] experience with three cases of macrocheilia of the upper lip in which pathological examination of tissue removed at operation showed the enlargement to be due to increase in size and enlargement of the labial salivary glands, which led him to conclude that this condition is more common than formerly believed, and that many cases of this type are erroneously diagnosed as lymphangioma.

Enlargement of the lip, when associated with characteristic ulceration, may be diagnosed clinically as in lues, carcinoma or tuberculosis. In the absence of ulceration, however, the diagnosis becomes more difficult. Macrocheilia without ulceration may be due to simple hyperplasia of the submucous salivary glands, to lymphangioma or lymphangiectasis, to elephantiasis or to hemangiomata. The angiomas may be differentiated by the compressibility of the swelling, its increase in size when the head is placed in a dependent position, the

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presence of varicosities beneath the surface of the skin or mucous membrane, and by the usually asymmetrical contour of the swelling. True elephantiasis is characterized by the appearance of thickened and hypertrophic folds of the skin and mucous membrane. Macrocheilia due to hyperplasia of the labial salivary glands is frequently confused with lymphangioma in the same region. Both conditions present a smooth, diffuse, nonedematous, nonulcerative, painless enlargement of the lip.

The literature on the subject reveals considerable confusion in nomenclature. The term macrocheilia has been applied to many cases in which there was no pathological report, but the condition was diagnosed lymphangioma. Cases of simple hyperplasia of the labial glands have been reported as adenomas of the lip, although no evidence of an encapsulated tumor was present. Double lip and its operative treatment have been reported but with meager mention of its pathology. Macrocheilia associated with elephantiasis and enlargement of the thyroid gland was described by Ascher[6] and believed by him to be a new clinical entity. This frequently is referred to as Ascher's syndrome.[7] Under the title of cheilitis glandularis apostomatosa or myxoadenitis labialis, Volkmann[8] described as macrocheilia a lesion characterized by ulcerations representing muciparous glands with dilated follicular orifices which admitted a fine probe and from which mucoid, mucopurulent or serous fluid could be expressed.

New and Kirch[4] report 67 cases of permanent enlargement of the lips and face secondary to recurring swelling, and 13 of these were associated with facial paralysis. They believe this to be a new clinical entity. The biopsy reports of their cases are very meager. They state: "Specimens of tissues were taken for biopsy in a few cases before treatment, but nothing was found other than edematous tissue containing lymphocytes."

Touraine and Solente[9] stress the precancerous nature of the condition, which they name cheilitis glandularis. One of the three cases described by Conway[5] presented a "double lip"

appearance. This patient developed mucus fistulas of the type described by Volkmann.[10] Another had recurrent swellings followed by permanent enlargement of the lip associated with facial paralysis. All these cases showed the same underlying pathological changes in the tissue of the enlarged lip; namely, hyperplasia of the labial salivary glands. In one case there was an associated paralysis of the facial nerve. Microscopy revealed, in addition, a diffuse inflammation of the submucous tissue. Conway[5] prefers to classify these three cases as "hyperplasia of the labial salivary glands" rather than the older term, "cheilitis glandularis," recognizing that the hyperplasia may occur either with or without evidence of inflammation. Kettel[11] describes a condition known as Melkersson's syndrome, consisting of *1.* peripheral facial palsy, which may be familial and which may show a tendency to relapse, *2.* angioneurotic facial edema, and *3.* lingua aplicata.

HISTORICAL.

Inflammation of the lips, or cheilitis, has been described as early as 1682 by C. Stalpartius Van der Wiel.[12]

Bouisson,[13] in 1869, referred to tumors of the labial salivary glands and states that, as yet, these conditions have not been subjected to precise observations. In 1870, Volkmann[8] gave the name of cheilitis glandularis apostomatosa or myxadenitis to a syndrome which he had observed in five cases, three of which had syphilis. The lower lip was affected in all five. On examination these cases showed that the salivary glands of the lip had become "hempseed size," were swollen and were palpable as uneven masses in unusual number. On everting the mucosa of the lip, the excretory ducts appeared so dilated that a probe could be inserted. On moderate pressure an opaque mucous or purulent secretion could be expressed.

The acute circumscribed swelling described by Quincke,[14] in 1882, corresponds with the onset of macrocheilia, but Quincke did not mention the residual enlargement. In Quincke's edema,[14] although the swelling has a preference for certain regions, such as the eyelids, cheeks, lips and hands, it does not always recur in the same area.

Wright,[15] in 1884, described a case in an 18-year-old boy who had a swelling of the lip which had been present since birth. The glands were palpable beneath the intact mucosa. Successful removal was performed by a two-stage operation. Microscopic examination was not made.

Prior to the discovery of the spirocheta pallida and the serologic reactions for syphilis, and before the advent of modern therapy for lues, many considered syphilis one of the prinicipal causes of elephantiasis of the lips. Macrocheilia was frequently associated with syphilis. In 1888, Mracek and Long[16] described a chronic edematous condition of the lips.

In 1892, Miles[17] reported a case of enlargement of the upper lip, which on gross examination showed the labial glands to be hypertrophied. The same year, Fraenkel[18] attached the name macrocheilia to an enlargement of the lower lip due to an adenomatous tumor of this region. The gross specimen was described as a "pearl-like string of tumors." This occurred in a 19-year-old boy and had been present since the age of four years. Hübschmann,[19] in 1894, and Rossolino,[20] in 1901, reported some cases in which, besides relapsing facial palsy, there was transient facial edema involving the lips.

In 1901, Tourniére,[21] under the title of diffuse hypertrophic syphiloma, described macrocheilia as syphilitic elephantiasis.

In 1904, Eisendrather[22] reported the case of a 16-year-old boy who had swelling of the lips since the age of 10 years. Microscopical examination of tissue removed showed "normal labial mucous gland substance." There was associated ptosis of both eyelids.

In 1905, Lindenberger[23] described the case of a man in whom the swelling followed a blow received on the lip 15 years earlier. Gross pathological examination revealed a number of small glands "slightly larger than buckshot." No microscopic examination was made. About the same time, Donati[24] described the same condition in a 20-year-old man. The swelling of the upper lip had been present for eight years. The excess tissue was excised and tissue examination showed it was due to enlarged mucus glands. Roussel,[25] in 1906, reported a case

in which the lower lip had been swollen for three years. Mucus fistulas and luetic skin lesions were also present. Microscopic examination was not made.

In 1911, Masera[26] reported two cases. One was in a woman of 24, the other a youth of 19. Pathological examination showed the tissue to be composed chiefly of acinous labial glands with many excretory ducts. This writer believed the macrocheilia to be due to hypertrophy and hyperplasia of the labial mucus glands.

In 1915, Stelwagon[27] suggested that the enlargement of the lip was due to adenoma of the mucous glands of the lip, and was not due to syphilis.

In 1920, Ascher[6] described a case presenting the syndrome of blephrochalasis, struma and double lip. Wirth[28] presented a similar case with lid changes and double lip without enlargement of the thyroid gland. In 1920, Sutton[29] described a case of macrocheilia of the upper lip due to ademnoma of the mucus glands in a man of 26. Microscopic examination showed numerous mucus glands.

In 1921, Weve[30] reported a patient with Ascher syndrome in a case of a 27-year-old healthy soldier who showed blephrochalasis and double lip. Both had been present since the patient was four years old.

Ascher (1922) described two additional cases of the same syndrome. One was in a 16-year-old girl, the other a man of 39 who had swelling of the eyelids, double lips and painless swelling of both parotid glands.

Dorrance[31] (1922) reported a case of double lip of nine years' duration in a patient 21 years of age. Gross examination of the specimen from the upper lip revealed enlargement of the labial salivary glands. Microscopic examination was not reported. In 1922, Roedelius[32] described a case of macrocheilia of the lower lip with several sinuses which admitted a probe and through which mucus discharged. This patient was a 45-year-old man. The enlargement of the lip had been present since childhood. In infancy this patient had had an operation

for harelip. Roedelius advanced the opinion that in patients with congenital harelip the protrusion of the lower lip might be a factor in exciting activity of the mucus glands, thus adding to their overgrowth. In 1928, Melkersson[33] presented his syndrome of *a.* peripheral facial palsy, *b.* angioneurotic facial edema, and *c.* lingua plicata. In 1930, Myer[34] reported two cases — one in a 10-year-old boy, the other in a woman of 36. This was associated with marked abnormality in the shape of the alveolar process of the upper jaw in both cases. Histlogical examination showed the membrane, the labial glands and the surrounding tissues to be normal.

Rosenstein[35] (1932) reported a case of Ascher's syndrome in a 16-year-old girl. In this patient there was enlargement of both lips, a tendency to hyperthyroidism and enlargement of the eyelids. Because of the enlargement of the lips developed at the onset of menstruation, Rosenstein believed the macrocheilia to be due to disturbance of the endocrine glands. In 1933, Bejarano[36] reported two cases of cheilitis glandularis associated with epithelioma. In both cases the lower lip was involved. The author does not believe that the cheilitis was a genetic factor in epithelioma of the lip.

Touraine and Solenter[37] (1933-1935) described two cases and reviewed 41 cases from the literature. In both of their cases the lower lip was involved; one was of six months' duration in a woman of 60. In the former, a spindle cell epithelioma was found at the base of the infundibulum. Out of the 41 cases in the literature they reviewed, 12 per cent showed malignant changes and were considered by them as precancerous.

In 1933, Eitner[38] reported nine cases, of which three showed enlargements of the eyelids, in addition to the double lips, but none exhibited an enlargement of the thyroid gland. The histopathological examination was not given. In New and Kirch's[4] 61 cases mentioned above, the swelling of the face was distributed as followed: Upper lip, 38 cases; nose, four cases; upper alveolar process, two cases. In their report the authors make no mention of Ascher's cases, nor do they com-

ment on the simultaneous involvement of the lips and eyelids in the same patient. As stated above, their pathological report is very meager. Conway[5] is of the opinion that many cases of macrocheilia are due to hyperplasia of the labial salivary glands. In discussion of the presentation of New and Kirch, Beck[39] mentioned three cases, two of which had a history of facial paralysis. Kettel[11] states that in three of four cases macroscopic swelling was due to edema and chronic inflammation but not due to lymphatic stasis. Dilated lymphatic vessels could be seen only in Case 4. Burkett[11a] (1946) reported a case of macrocheilia due to repeated streptococcal infections of the gums and lips.

Incidence: It is apparent that in most instances this condition (swelling of the lips and face) occurs mostly in the young. Females appear to be less prone to the disease than males. Many of these cases are associated with facial paralysis. Conway[5] reported three cases, with involvement of the facial nerve to one. New and Kirch,[4] out of 67 cases, 13 per cent showed facial paralysis. The ages of their patients at the occurrence of the first swelling ranged from four to 54 years. Twenty-nine patients were less than 20 years of age, and 50 were less than 30 years. The incidence by decades was as follows:

One to 10 years, three cases; 11 to 20 years, 26 cases; 21 to 30 years, 21 cases; 31 to 40 years, 10 cases; 41 to 50 years, five cases, and 51 to 60 years, two cases. There were 46 males and 21 females. Thus it may be seen that the condition occurs predominantly in youth and in patients less than 30 years of age. Males predominate over females in the ratio of better than two to one.

ETIOLOGY.

The cause of the condition is not known. Its similarity in some respects to angioneurotic edema has been noted. In angioneurotic edema there is involvement of other parts of the body than the face at the same time that the face is affected, but in the cases described above, only the face, mouth

and throat were involved. The common condition of edema as generally observed causes tenseness of the skin and pitting on pressure, but the residual swelling in these cases is soft and elastic. There are no changes in the skin itself.

The causative factors of this condition are purely theoretical. New and Kirch state that one must consider, first, a disturbance of trophic, vasomotor or motor nerves; second, alterations in the metabolism or state of nutrition of the tissues, changing the power of the cell to attract, give off or retain fluid; third, some chemical disturbances in the cell of a complicated nature that changes the fluid balance, and fourth, a possible so-called vital secretory activity of the endothelial cells. Kettel is of the opinion that the facial palsy, as well as the swelling of the face, is an angioneurotic phenomenon and the etiology must be sought in these factors which may cause a disurbance of the regulations of the vasomotor nerves.

TREATMENT.

New and Kirch advise the injection of boiling water, followed by radium application. Three months later, this treatment may have to be repeated. When there is no tendency to recurrence of the swelling for six months, the superfluous tissue of the lips or cheeks may be excised. They state that it is essential, first, to eliminate the recurring swelling before attempting operation in order to prevent recurrence or prevent greater enlargement of the parts than was present before operation.

Kettel performed excision only in Case 1, and the result was satisfactory. In Case 5, despite preceding injection of boiling water and subsequent partial excision, there occurred another attack of acute edema, which, however, subsided. Conway[5] reports the same experience. From the above, one must conclude that excision may be done without preceding injection of boiling water or radium treatments, running the risk of recurrent swelling, as the treatment referred to by no means protects the patients against relapse. Just how this

treatment stops the swelling has not been explained. It may be that the production of fibrosis of the lymph structures or changes in the cells themselves prevent swelling of the tissues; however, the results of this treatment have been satisfactory.

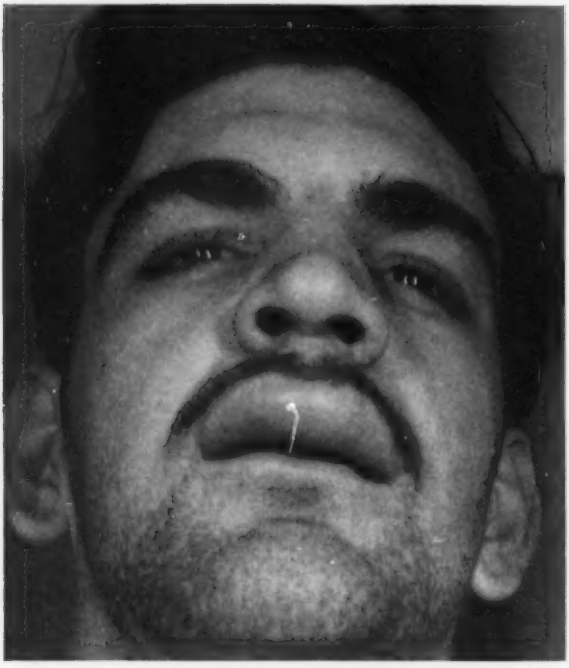

Fig. 1. Appearance of patient on July 15, 1946, following the injection of boiling water and prior to Roentgen and surgical treatment.

REPORT OF A CASE.

Male, aged 24. Hospital No. 1,211,596. Admitted to hospital on May 27, 1946.

C. C.: Swollen lip.

P. I.: The swelling of the upper lip appeared approximately 10 years ago. The swelling, which was never painful or tender, would occur about every three months, last from two to three days and disappear spon-

taneously; however, the swelling never completely disappeared, so that after each episode of swelling the lip remained a little larger than before. For the past year the swelling receded but very little. The present swelling came on four months ago and has so remained.

P. H.: Does not recall having any childhood diseases. At the age of two years had fractured femur. At the age of 12, patient developed

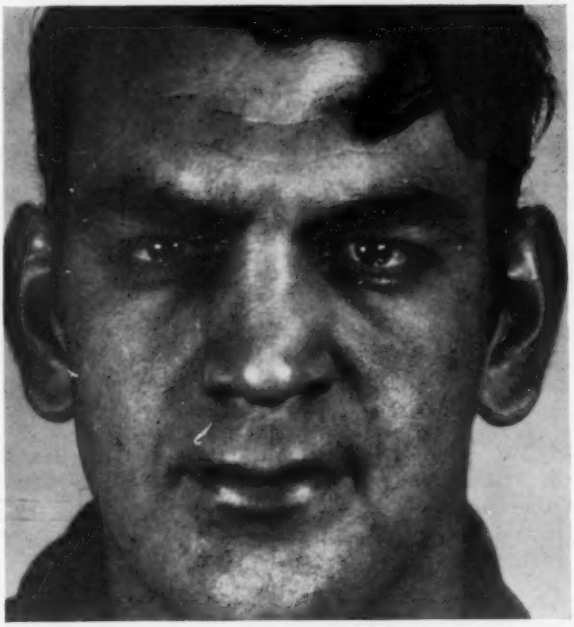

Fig. 2. Appearance of the patient at present, approximately two years following treatment.

poliomyelitis and was in the hospital for several years. Patient had an appendectomy operation four years ago and a tonsillectomy one year ago.

P. E.: Revealed a diffuse enlargement of the entire upper lip protruding markedly over the lower lip. The swelling was not tender or painful and did not feel edematous. On digital pressure the lip felt rather firm and in places "shotty." The lip was dusky red in color and there were no ulcerations, fissures or fistulas. Repeated serologic and urine examinations were entirely negative.

TREATMENT.

May 28, 1948: Under pentothal anesthesia, 15 cc. of boiling water were injected throughout the upper lip. After the reaction had subsided the patient was referred to Dr. Frederick W. O'Brien, who is in charge of the tumor clinic and who carried out the Roentgen therapy. He was kind enough to supply us with details of this treatment, which was as follows:

"Re: A. E., Tumor Clinic, Boston City Hospital. Referred to the tumor clinic July 15, 1946, by Dr. Garfin, with diagnosis of lymphedema. Had had injection of upper lip with boiling water one month before without appreciable reduction in size of lips.

"Patient was given 2,000 r high voltage X-rays, with 1.13 mm. cu., in divided doses, 7 cm. field, between July 15, 1946, and Aug. 5, 1946, at which time there had been an appreciable reduction in the size of the upper lip."

Operation was carried out on Nov. 4, 1946. Under novocaine anesthesia a wedge-shaped section of tissue was removed from the mucus surface of the upper lip down to the orbicular oris muscle. Bleeding points were ligated with fine catgut sutures, and the edges of the wound were approximated with one-end fine silk mattress sutures. Following the operation, patient was put on penicillin.

Pathological report was as follows:

"There is slight hyperkeratosis of the epithelium. There is an increase of the submucosal fibrous tissue with large amounts of collagen. There is moderate congestion of the blood vessels. No edema of tissue is seen. There is marked interstitial and perivascular infiltration with small round cells, macrophages and occasional polymorphonuclear neutrophilic leucocytes and plasma cells. There is an apparent decrease in striated muscle fibres."

Diagnosis: Chronic inflammation.

Over two years have elapsed since the treatment was started, and except for one episode of swelling which followed the X-ray treatment, the swelling of the lip gradually receded and has remained entirely normal without any recurrent swelling during this period.

SUMMARY.

1. A report of a patient with macrocheilia has been presented.

2. A review of the literature on macrocheilia reveals considerable differences of opinion as to the etiology and pathogenesis of this condition. It is apparent that the pathogenesis is not the same in all cases of chronic enlargement of the lip. In our case it was due to chronic inflammation.

3. The injection of boiling water followed by Roentgen therapy and surgical removal of excess tissue proved successful.

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AMERICAN BOARD OF OTOLARYNGOLOGY.

The American Board of Otolaryngology will conduct the following examinations in 1949:

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Oct. 4 to 7 in Chicago, Ill., at the Palmer House.

A SIMPLIFIED METHOD OF OBLITERATING FRONTAL BONE DEFECTS.*

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Reconstruction of the frontal area of the skull, following accidental trauma or operative procedures, has been described so often that one hesitates to comment further upon the subject.

A complete review of surgical attacks devised and materials used to efface bony defects in this region would be far too unwieldy for a brief presentation. In general, substances used fall into three main groups:

1. Autografts (*e.g.*, bone, cartilage, fascia lata, or derma removed from the patient's own body).
2. Homografts (*e.g.*, preserved rib cartilage in strips or small fragments).
3. Various nonanimal products (*e.g.*, vitalium, tantalum and acrylic).

Although different metals and other synthetic products have been widely used and highly praised, we have questioned the advisability of using foreign materials in the human body no matter how nonirritating they seem. Occasionally we have seen patients who have tolerated such implants poorly. Some have been in the process of extruding them, with resulting inflammation of the overlying soft tissue. Some have suffered psychic disturbances because of the knowledge that they were carrying extraneous substances in their skulls. On such occasions we have regarded these materials with even greater timidity.

In a lesser degree, we have questioned the desirability of cartilage homografts.

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Our choice of autografts is reduced to two skeletal tissues, bone and cartilage. Of the two, our choice is overwhelmingly in favor of bone. Our choice of bone, to be used whenever feasible, will be questioned by those who favor cartilage just as strongly; our adoption of cancellous bone is probably influenced by personal preference, but over the years we have been increasingly impressed by its favorable properties. We regard cancellous bone highly because of its availability in large amounts, its workability, and the almost certain assurance of firm fixation after transplantation.

Once the decision to use bone as a graft is made, it may be employed in one of three forms:

1. As a single plate.
2. As strips.
3. As chips of moderate size (1 cm. or larger).

When a large skull defect is present it is often difficult to find a plate of bone large enough to fill it, and one which may be pared to the proper contour; in addition, there is usually the problem of preventing a dead space beneath the graft. This issue is also present when curved strips are used, and further trouble is encountered when one tries to fit the strips together in such a way that the forehead will not have a corrugated surface.

The use of chips to fill a defect is technically less demanding than the modeling of a plate or strips, but if they are of appreciable size, and especially if every chip is in contact with the recipient bed, it is difficult to arrange them so that a smooth and convex surface results.

Obviously, the simplest way to achieve a proper bony contour would be to cut the osseous graft into extremely minute particles and insert them into a previously prepared pocket — much like "stuffing feathers into a pillow." The amount and distribution of this easily molded material could be determined by external manipulation, and should result in a surface of any desired shape.

Two questions immediately arise concerning the use of

bone about the skull in such a manner: will the resulting bulk of bone have a deleterious effect on the intracranial contents because of direct irritation and/or pressure? Will the small particles of bone in the center of the transplanted mass remain viable long enough for a blood supply to be derived from the peripheral recipient bed?

In regard to the first question, we consulted Dr. Russell Meyers, of the Division of Neurosurgery, University Hospital, who felt that no damage to the brain would result from such

Fig. 1.

a procedure, especially if the graft could be laid on top of the greatly thickened dura that results from a healed inflammatory process.

In answer to the second question, Dr. Arthur Steindler, of the Department of Orthopedic Surgery, assured us that we need have little fear of losing the deep particles of the graft because of inadequate blood supply. He not only felt that adequate nutrition would be obtained, but was convinced that

all the tiny fragments of bone would be solidified into one solid mass within a few weeks.

These encouraging and authoritative answers to our most obvious problems led us to attempt the obliteration of large frontal bone defects with bits of cancellous bone cut as small as possible. We were amazed at the ease with which reconstruction could be carried out, and at the satisfactory results obtain from such a relatively simple procedure.

Two illustrative cases are presented.

Case 1: G. J., No. 45-834, a 36-year-old white male, was first admitted to the University Hospitals on Aug. 27, 1945, with a diagnosis of acute purulent frontal sinusitis (bilateral), complicated by fulminating acute osteomyelitis of the frontal bone. At operation it was necessary to sacrifice

Fig. 2.

both frontal sinuses in addition to the portion of the frontal bone forming the center one-half of the forehead. The patient had an uneventful postoperative recovery and was observed for 28 months to make certain that no residual infection was present.

On Dec. 9, 1947, the patient was readmitted for obliteration of the frontal bone defect depicted in Fig. 1.

At operation a large amount of cancellous bone was removed from the posterolateral surface of the ilium. The removed bone was then cut into

pieces (2 to 4 mm. in diameter) as small as possible with heavy suture scissors.

A coronal incision in the hair-bearing area of the scalp, about 4 cm. above the edge of the defect in the frontal bone, was carried down to the periosteum. Through this incision the scalp was separated from the greatly thickened dura in the area of the defect by blunt and sharp dissection; elevation was carried subperiosteally over bone about 2 cm. beyond the edges of the depression. The previously morcellated cancellous bone was introduced into the pocket thus formed. By external molding during introduction of the graft, the correct contour of the forehead was easily produced.

Six weeks after operation, the graft was solid to palpation and presented the appearance shown in Fig. 2. X-ray examination was reported "... showing the previous defect in the frontal bone to be filled with bony tissue; it appears that the graft is viable and that healing is in progress."

In the 11 months that have elapsed since operation, we have no report of any further changes in the graft.

Case 2: R. M., No. 44-5460; a summary of this case would be almost a repetition of the first one. It is presented only in order to call attention to a variation in operative technique that made the procurement and morcellation of the cancellous bone much simpler.

In this instance the anterior one-half of the crest of the ilium was exposed and cancellous bone was obtained from between the inner and outer plates by means of a small curette.

DISCUSSION.

It is felt that autogenous cancellous bone is the material of choice to be used in the correction of depressed areas of the frontal bone. The practice of implanting the bone in minute bits offers a method of shaping the graft to the proper contour without resorting to the extensive soft tissue dissection needed to place correctly a laboriously fashioned graft of large size.

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EDEMA OF THE UVULA FOLLOWING DEMEROL-HYOSCINE ANALGESIA.*

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A review of the otolaryngologic literature for the past 10 years has failed to reveal a case report or discussion of edema of the uvula due to hyoscine medication. Although this seems to be a problem encountered primarily by the obstetrical service, where hyoscine or scopolamine is used in a large number of cases for analgesia, in several of the eight cases reported in the obstetrical literature an otolaryngologist was called to assess the cause of acute upper respiratory distress in a patient in active labor.

Kirschbaum[1] first referred to edema of the uvula, in 1942, in a discussion of scopolamine in obstetrics and reported that occasional enlargement of the uvula to five or six times normal size was observed. Steinberg,[2] in 1945, reported four cases of edema of the uvula and glottis as a reaction to demerol-scopolamine analgesia, and one case required tracheotomy as a life-saving measure. The other cases responded to scarification of the uvula and local astringents. Skin tests in his four cases with 1-1,000 gr. of sopolamine intradermally were all positive in varying degrees. In 1948, Jack and Taylor[3] reported in detail one case of edema of the uvula and glottis that required tracheotomy, and three additional cases that responded to lesser measures. Since various other drugs had been combined with scopolamine, it was the consensus of opinion of the authors that scopolamine was the offending drug in all of the cases.

This case of hyoscine sensitivity which caused edema of

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the uvula and respiratory obstruction is reported as an otolaryngologic problem of acute upper respiratory distress encountered on the obstetrical service.

A 31-year-old white primigravida at term entered the obstetrical service of the St. Louis City Hospital on Oct. 11, 1948, in active labor. She was a pre-eclamptic with a history of ankle edema of two months' duration, blood pressure of 150/100 and urinary findings of many casts and three-plus albumin. Her prenatal course otherwise had been uneventful. On admission at 11:15 A. M., she received 100 mg. of demoral and 1-150 gr. of hyoscine. Her labor continued periodically through Oct. 11 and 12. Clinically, her toxemia had subsided and was well under control. On Oct. 13 at 10:15 A. M., she received 50 mg. of demorol and 1-150 gr. of hyoscine. At noon the patient developed aphonia and marked respiratory difficulty. An otolaryngology consultation was requested and examination revealed a cyanotic, semistuporous patient having marked inspiratory distress. The otolaryngologic examination was essentially normal except for a large grayish-white, tense, polypoid, midline mass which filled the entire pharynx. When the mass was lifted out of the pharynx, it was seen to be the uvula which was enlarged approximately 10 times its normal size. The patient was given ephedrine sulphate ⅜ gr. intramuscularly and placed in a Burgess box. Tracheotomy and emergency bronchoscopy sets were placed at the bedside. There was no diminution of size of the uvula after administration of the ephedrine, although the cyanosis decreased somewhat, and the respirations were less labored after the patient had been placed in the Burgess box.

Due to the lack of fetal heart beat and the patient's general condition, the obstetrical service decided to deliver the patient of the nonviable fetus by forceps. Since pentothal anesthesia was to be administered, it was felt that the danger of laryngospasm could be reduced by topical anesthesia of the pharynx and larynx. This was secured with 2 per cent pontocaine solution topically. During the induction with pentothal the uvula was lifted from the pharynx with a forceps to give a better airway. After surgical anesthesia was reached, stridor was still present; therefore, the Jackson laryngoscope was passed and thick mucoid secretions removed from the glottic chink by suction. The interarytenoid mucosa and false cords were somewhat pale and edematous; the true cords, however, appeared to be normal. An airway was inserted and the patient tolerated the forceps delivery and pelvic repair without further respiratory difficulty.

The postoperative course was uneventful; the tracheotomy and bronchoscopy sets were kept at the bedside, but were not needed. On the following day the uvula had diminished in size by one-half, and on the third postoperative day it appeared to be of normal size but still somewhat reddened.

On Oct. 27, an intradermal skin test with hyoscine was done. The control showed no reaction and the test area showed 4 mm. of erythema. The patient was discharged from the hospital on Oct. 28.

SUMMARY.

A summary of the literature of edema of the uvula and glottis following hyoscine (scopolamine) analgesia and the ninth such case to be reported have been presented. The occur-

rence of edema of the uvula and glottis following hyoscine is infrequent; however, the treatment must be prompt and adequate to control the respiratory embarrassment. In the cases reported in the literature this was obtained by tracheotomy, scarification of uvula, or by the application of local astringents. In this case the obstruction was relieved by lifting the uvula out of the pharynx, topical anesthesia and the insertion of an airway. Tracheotomy and bronchoscopy sets should be immediately available if needed.

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CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

On Nov. 12, 13 and 14, 1948, the Central Illinois Society of Ophthalmology and Otolaryngology held their eleventh meeting at the Abraham Lincoln Hotel in Springfield, Ill. Dr. F. Bruce Fralick, professor of ophthalmology at the University of Michigan, spoke on "Surgical Anatomy of Operations for Glaucoma" and "Diseases of the Tear Duct." Dr. F. L. Lederer, professor and head of the Department of Otoloryngology, University of Illinois, discussed "Modern Approaches to the Prevention and Treatment of Defective Hearing" and "Malignancy in Its Relationship to Nasal Obstruction and Hoarseness." Dr. L. W. Roth, Belleville, Ill., gave a paper on the "O'Connor Cinch Operation," and Dr. D. K. Judd, of Kankakee, Ill., discussed "Malignancies of the Paranasal Sinuses." Thomas W. Samuels, an attorney from Decatur, Ill., gave a delightful talk on "A Lawyer Looks at the Medical Profession." The following officers were elected for 1949: President, Dr. Clifton S. Turner, Peoria, Ill.; President-elect, Dr. Harold Watkins, Bloomington, Ill.; Vice-President, Dr. Meredith Ostrom, Rock Island, Ill.; Secretary-Treasurer, Dr. Philip R. McGrath, Peoria, Ill.

REPORT OF A CASE OF HERPES LARYNGIS.*†

DANIEL B. MOFFETT, M.D., and JAMES J. MCFARLAND, M.D.,
Washington, D. C.

Herpes zoster was well known to the ancient clinicians, having been commented upon in the time of Hippocrates. To the Greeks it was known as "zoster"; the Romans called it "zona" and "cingula," hence the Latin and French word, "chengle," from which is derived our English word, "shingles."[1]

The disease is a virus infection of the posterior root ganglia, and is thought by some to be similar to varicella virus. Hunt[2] calls it acute posterior poliomyelitis. The specific virus has never been isolated.

Herpes laryngis can be classified as rare; Landon[3] states that only 26 cases have been reported in the literature. Our review of more recent articles failed to disclose any additional reports. Hunt,[4] however, believes that it is more frequent than is commonly supposed. There are many diseases involving ulceration and exudation surrounding the mucous membrane of the throat and larynx, which might possibly be confused with herpes laryngis. In all cases reported, however, the picture of herpes laryngis was quite definite and unmistakable.

Pathologically, it is very similar to anterior poliomyelitis in that round cell infiltration of the posterior root ganglion is found. This is followed in many cases by acute hemorrhage into the ganglion, with secondary scar formation and possible permanent damage. In the lesions of the mouth and throat the pathology is always unilateral.

Each nerve has a characteristic intraoral, pharyngeal and

*Case presented before the Staff of the Episcopal Eye, Ear and Throat Hospital, Washington, D. C., Nov. 5, 1948.

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laryngeal zoster zone. We are not concerned here with eruptions of the external skin, except those of the external ear. Those involving the mucous membranes of the mouth and pharynx, the external ear and auditory canal have been defined by Hunt[5] as follows:

The intraoral, pharyngeal and laryngeal zoster zones of the vagus cover the epiglottis to midline, the arytenoid eminences, the aryepiglottic folds, the pyriform sinus, a wedge-shaped area at the base of the tongue in front of the epiglottis and a portion of the soft palate. The Xth cranial nerve produces skin manifestations of a posterior medial strip of the auricle and immediate adjacent mastoid area and the posterior portion of the drum and auditory canal; however, the auricular zoster zone of the Xth cranial nerve cannot be distinguished from that of the IXth cranial nerve.

The zoster zone of the IXth cranial nerve includes the posterior margin of the soft palate, anterior and posterior tonsillar pillars. tonsil and the posterior lateral surface of the base of the tongue.

The zoster zone of the VIIth cranial nerve is manifested by facial paralysis and cutaneous eruption of the concha, antihelix and fossa of the antihelix.

The zoster zone of the Vth cranial nerve involves the anterior two-thirds of the tongue, plus the labial and gingival mucosa (skin manifestations excluded).

It is interesting to note that, although the vagus nerve supplies the larynx completely, the eruptions of the mucous membrane rarely, if ever, involve the false or true cords.

During the first 48 hours of the acute stages of herpes laryngis, there is nothing more than slight inflammation. Vesicle formation follows within a short time. The vesicles at first contain clear serum, but this soon becomes turbid and is replaced by a brownish-yellow purulent fluid.

During the next stage of the disease, the vesicles rupture, leaving shallow ulcers with bright red areolae. At first the

ulcers are discrete but later they become confluent and are covered with a gray-white membrane which, when removed, often leaves a bleeding surface.

Edema of the epiglottis and arytenoid on the involved side, and edema of the uvula, are common findings. There is usually cloudy spinal fluid, with lymphocytosis. Encephalitis may be a complication of the disease.

The aryepiglottic fold, arytenoid cartilage and epiglottis are the sites of predilection. The true cords and ventricular bands, as a rule, are not involved except for a slight inflammatory reaction. Usually the pyriform sinus and lateral wall of the pharynx remain clear; occasionally the base of the tongue is involved. It is rare for the vesicles to appear on the larynx first, and still rarer for the larynx alone to be involved.

Recurrences are most uncommon and, since this is true, a careful check of the patient's past medical history is often helpful in making a diagnosis. Predisposing factors include colds and influenza, chronic nervous disorders, chronic foci of infection and other virus diseases.

The disease process usually terminates in one or two weeks, ending in complete recovery and no permanent sequelae. In the beginning of the illness the patient commonly suffers mild chills, moderate fever, general malaise and a burning, scratching sensation in the throat, with severe pain on swallowing. With the appearance of the vesicles, the symptoms are intensified. Extreme pain in the throat, particularly when swallowing, is the most prominent symptom. A slight hoarseness is common. After the rupture of the vesicles, the symptoms become less severe but may persist for some time.

Neural complications may appear singly or in various combinations, depending on the ganglion or ganglia involved. Nausea, vomiting, hiccoughing, bradycardia, etc., may appear as a result of the involvement of sundry branches of the vagus nerve. These neural symptoms are often transient, disappearing in a few days; on the other hand, they may last a fortnight or longer. The virus infection can easily spread from one cranial ganglion to another, and there may be involvement

of the ganglion of the Xth, IXth, VIIth or Vth nerves, with or without facial nerve involvement. The resulting facial paralysis may be permanent. Postherpetic signs such as burning sensation, coughing, hiccoughing and neuralgia may last for many months or even years.

There is no specific treatment for herpes zoster. Adequate diet, bed rest and soothing applications are helpful. Injections of snake venom, pituitrin, X-ray, vaccination with blister fluid or smallpox have all been tried with varying degrees of success. Despite the lack of a specific remedy, the prognosis is good. None of the cases reported has been fatal and, as mentioned before, the possibility of recurrence is rare. It should be remembered, however, that all cases of herpes laryngis reported have been unilateral.

CASE REPORT.

E. G., white female, aged 58 years, was first seen in our office on July 19, 1948. She complained of extreme soreness and tenderness of the left side of the throat, with excruciating pain and burning sensation in the left pharynx, radiating to the left ear. She had some nausea and difficulty in swallowing. Her temperature was 100° F.

The patient's past medical history was not significant. Her family history revealed that a sister had "shingles" in 1936.

Examination showed nothing abnormal other than a very slight hyperemia of the lateral bands of the left pharyngeal wall. The ears were entirely negative. Although she complained of some hoarseness, no pathology of the vocal cords was noted.

Local treatment was ineffectual, and she became progressively worse.

On July 21, she was admitted to the hospital. At this time her temperature ranged from 101.5° to 102° F.

On July 22, small blisters appeared on the soft palate and the left lateral pharyngeal wall, posterior to the left tonsillar fossa. Subsequently, the blebs ruptured and formed white, shallow ulcers which were covered with necrotic membrane and surrounded by aerolae of inflammation. At first they were discrete, but later became coalescent and eventually involved the left side of the soft palate, the pharyngeal wall, the epiglottis to the midline and the aryepiglottic fold. They stopped short, with a sharp line of demarcation, just above the level of the false vocal cords. Although she continued to have some hoarseness, the true cords were never involved; there was rather marked edema of the left arytenoid, but no involvement of its mucous membrane, nor was there any limitation of motion.

Throat culture revealed streptococcus viridans, and was negative for diphtheria. Urinalysis was negative. Blood analysis showed a hemoglobin of 75 per cent; R. B. C., 4,000,000; W. B. C., 6,400; 79 per cent polymorphonuclears; 20 per cent lymphocytes; N. P. N., 25 mg. per cent.

She was given penicillin, 40,000 units of diphtheria antitoxin and intramuscular injections of large doses of thiamin chloride. Her nausea and vomiting became more pronounced, for which she was given intravenous glucose. At this time she developed paroxysms of coughing which continued all the time she was in the hospital.

On July 28, after seven days' hospitalization, she was discharged. Her temperature had returned to normal; the ulcers and whitish membrane of the pharynx and larynx had disappeared. Some referred pain in the left ear persisted, and she still complained of inability to swallow food and difficulty in retaining it afterwards.

On Aug. 5, she was readmitted to the hospital because of a marked increase in the gastrointestinal disturbances. She had severe nausea and vomiting with pain in the stomach. She could not eat or drink anything except small amounts of barley broth.

Her coughing paroxysms continued, brought on by what the patient described as "something on the back of my tongue." She also developed hiccoughs which continued, intermittently, for two days. Each attack of hiccoughing lasted for approximately 30 minutes.

On Aug. 13, she was again discharged from the hospital, her gastric symptoms having subsided. The pain in the throat and ear persisted, but was much less severe.

On Sept. 30, she was seen in the office. She now described a feeling "as if an electric current were running from the left ear to the larynx," a sensation which invariably brought on a paroxysm of strangling and coughing and which occurred about once every hour. Rough foods or alcohol aggravated the symptoms. We found that we could produce similar attacks by pressing on a trigger zone just behind the posterior tonsillar pillars on the left.

By Oct. 27, some three and one-half months after the onset of the illness, the tick disappeared. There were still occasional paroxysms of coughing, especially at meal time, but the other symptoms had subsided. She spoke of being bothered by watering of her left eye and by occasional blurring of vision.

At the present time, the patient appears well. All symptoms have disappeared, except some paroxysms of coughing at meal time. There is no apparent permanent damage as a result of her illness.

Of the various medications and treatments prescribed, we feel that she dervied most benefit from the massive doses of thiamin chloride.

SUMMARY.

Herpes zoster is a virus infection of the posterior root ganglia. Each nerve has a characteristic zoster zone. The case of herpes laryngis presented shows a typical pattern of herpes zoster of the left vagus nerve.

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3. LANDON, H. R.: Herpes Laryngis. *Arch. Otolaryngol.*, 10:16-20, 1929.

4. HUNT: Op. cit., p. 635.

5. *Ibid.*, p. 633-635.

1150 Connecticut Avenue.

MISSISSIPPI VALLEY MEDICAL SOCIETY 1949 ESSAY CONTEST.

The Ninth Annual Essay Contest of the Mississippi Valley Medical Society will be held in 1949. The Society will offer a cash prize of $100.00, a gold medal, and a certificate of award for the best unpublished essay on any subject of general medical interest (including medical economics and education) and practical value to the general practitioner of medicine. Certificates of merit may also be granted to the physicians whose essays are rated second and third best. Contestants must be members of the American Medical Association who are residents and citizens of the United States. The winner will be invited to present his contribution before the Fourteenth Annual Meeting of the Mississippi Valley Medical Society to be held in St. Louis, Mo., Sept. 28, 29, 30, 1949, the Society reserving the exclusive right to first publish the essay in its official publication — the *Mississippi Valley Medical Journal* (incorporating the *Radiologic Review*). All contributions shall be typewritten in English in manuscript form, submitted in five copies, not to exceed 5,000 words, and must be received not later than May 1, 1949. The winning essays in the 1948 contest appear in the January, 1949, issue of the *Mississippi Valley Medical Journal* (Quincy, Ill.).

Further details may be secured from Dr. Harold Swanberg, Secretary, Mississippi Valley Medical Society, 209-224 W. U. U. Building, Quincy, Ill.

A NEW SPECULUM FOR SEPTAL SURGERY.*

DANIEL D. KLAFF, M.D.,
St. Louis, Mo.

The illustrated instrument was designed to facilitate better visualization of the septum when performing the complete or classical submucous resection. The construction is simple and practical:

A spring controls the size of the aperture between the blades to facilitate the insertion of the instrument between the mucosal flaps. This opening may be diminished by pressing the blades together with the fingers. The tension produced by the spring is never great enough to tear the mucosal flaps. If further widening of the flaps is desired, it may be obtained by rotating the screw at the side of the shank. When the blades are parallel, the aperture is sufficient to permit a clear view of the septum, thus the instrument is self-retaining. There is no tunnelling inward of the blades toward the perpendicular plate of the ethmoid. Dorsal and posterior vomerine spurs may be clearly seen. The instrument is easily inserted, self-retaining and permits a clear view of the posterior portion of the septum.

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*From the Department of Otolaryngology, Washington University School of Medicine.

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THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

The annual meetings of the Triological Society and the Broncho-Esophagological Association will be held concurrently at the Drake in Chicago on April 18, 19 and 20, 1949. A joint meeting of these two societies will take place Tuesday morning, April 19. On the other days the Triological Society will meet in the mornings and the Bronchoscopic Society in the afternoons.

We also call your attention to these dates and places:

American Board of Otolaryngology—May 11-14, 1949, New York City.

American Laryngological Association — May 16-17, 1949, New York City.

American Otological Society—May 18-19, 1949, New York City.

In order to facilitate the coordination of national meetings, the Bronchoscopic, the Otological, the Laryngological and the Triological Societies are considering a five-year plan of dates and places. By adoption of such a scheme we hope to reduce the traveling mileage and the amount of time away from our offices. For additional information, write to Dr. C. Stewart Nash, Secretary, 708 Medical Arts Building, Rochester 7, N. Y.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

As of January 1, 1949.

Acousticon Model A-100.
Manufacturer: Dictograph Products Corp., 580 Fifth Ave., New York 19, N. Y.

Aurex (Semi-Portable); Aurex Model C-B, Model C-A, Model F and Model H.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac; Beltone Harmony Mono-Pac.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Type K; Maico Atomeer.
Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

1947—Mears Aurophone Model 98; Mears Aurophone Model 200.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver).
Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-4 Audiomatic; Microtone T-5 Audiomatic.
Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model; National Standard Model; National Star Model.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model A-1; Otarion Model A-3; Otarion Models A-4 J and S; Otarion Model E-1; Otarion Model E-1S; Otarion Model E-2.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL; Paravox Model XT; Paravox Model XTS.

Manufacturer: Paraphone Hearing Aid, Inc., 2056 E. 4th St., Cleveland, Ohio.

Precision Table Hearing Aid.

Manufacturer: Precision Electronics Co., 850 W. Oakdale, Chicago 14, Ill.

Radioear Masterpiece; Radioear 45-CM; Radioear Model 45-M-magnetic air conduction receiver; Radioear Model 45-M-magnetic bone conduction receiver; Radioear Permo-Magnetic Uniphone.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Ravox (Semi-Portable).

Manufacturer: Zenith Radio Corp., 6001 W. Dickens Ave., Chicago, Ill.

Silver Micronic Hearing Aid Model 101; Silver Micronic Hearing Aid Models 202M and 202C.

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

Solopak Hearing Aids.

Manufacturer: Allen-Howe Electronics Corp., 150 Main St., Peabody, Mass.

Sonotone Audicles No. 530, No. 531 and No. 533; Sonotone Model 600; Sonotone Model 700; Sonotone Model 900.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Telex Model 22; Telex Model 97; Telex Model 612; Telex Model 900; Telex Model 1020; Telex Model 1550.

Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.

Manufacturer: Tonemasters, Inc., 1627 Pacific Ave., Dallas 1, Tex.

Trimm Vacuum Tube No. 300.

Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A."

Manufacturer: Nichols & Clark, Hathorne, Mass.

Vactuphone Model 3.

Manufacturer: Allen-Howe Electronics Corp., Salem, Mass.

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Manufacturer: Western Electric Co., Inc., 120 Broadway, New York 5, N. Y.

Zenith Radionic Model A-2-A; Zenith Radionic Model A-3-A; Zenith Radionic Model B-3-A; Zenith Model 75.

Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

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